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BEHAVIOR PROBLEMS IN CHILDREN

HARRY BAKWIN, M.D.
NEW YORK

FOURTEEN years ago the late Dr. Joseph Brennemann addressed this society on "The Menace of Psychiatry." [1] With the wit and graceful phraseology of which he was master, he discussed the dangers of child psychiatry in the hands of amateurs. What he said fourteen years ago remains true today: There are still too many mothers playing psychologist to their children. Some experiences of mine along this line can, I am sure, be matched by those of any pediatrician.

Recently a mother called to ask a question about her 5 week old baby. The patient had been referred to me by a psychiatrically minded pediatrician who had entered the armed forces. The mother said that for the past three days her baby had been crying during the late afternoon. She wanted to know whether she had not started to socialize the baby a little too early.

Another mother stopped the breast feeding of her 6 week old baby—although the little girl was doing beautifully—because, the mother said, she had not nursed her first-born child and she was afraid that having nursed one and not the other of her children might be the basis for a sibling rivalry later.

As Franklin P. Adams [2] so aptly said, "Some mothers know their Jung better than their young."

It is highly gratifying to the pediatrician to review the amazing success of the program of physical care for children. Since the turn of the century—a span of less than fifty years—infant mortality has been reduced by three fourths. Fatal infantile diarrhea has been almost eliminated. Contagious disease has become a minor cause of death. The death rate from prematurity is being reduced. All along the line, physical disease is being attacked with a courage and tenacity that excite the highest admiration. Even for some of the congenital anoma-

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From the Children's Medical Service of Bellevue Hospital and the Department of Pediatrics, New York University.

1. Brennemann, J.: The Menace of Psychiatry, Am. J. Dis. Child. **42**:376 (Aug.) 1931.

2. Adams, F. P.: Proposition: Am I a Good Father? Esquire, July 1945, p. 34.

lies, hitherto looked on as largely beyond help, there is now some hope. Prominent among the bold whittlers away at death and disease have been the Boston surgeons Ladd, Ingraham and Gross.

In contrast to the vigorous campaign directed toward the maintenance of optimal physical health and the prevention of physical disease and death is the lack of a concerted program aimed at the promotion of optimal psychologic health and the prevention of disorders of the psyche. Though the more frequent recognition of "psychoneurosis" is in good part explained by the greater interest in this subject, there seems little doubt that disturbances of this nature are widespread and that they constitute a serious threat to the happiness—and even the safety—of society.

LIMITATION OF FAMILY SIZE

Some of the causes of psychoneuroses are outside the control of the physician. Prominent among these is the limitation of family size, with the resulting morbid concentration of parents on one or two children. Children in large families have an opportunity to learn to make the adjustments necessary for social living within the protecting boundaries of the home—an opportunity which is necessarily lacking in small families.

In addition, many women have an idea that motherhood and home building are, at best, incomplete outlets for their energies. They feel that they should be doing something worth while—by which they mean activities outside the home.

The small family is, increasingly, a reflection of a state of mind. There is abroad—and there has been for some years, even before the war—an altered attitude toward the values of life. America was built on courage and optimism and self sacrifice, on faith in a way of living and faith that our breed was worth preserving. But within recent years doubts have entered the minds of many, and the qualities which have made possible the enormous success of America have been replaced by fear and selfishness and pessimism. Such attitudes cannot but have an unfavorable influence on the developing child.

These widespread changes in attitudes are to a large extent beyond the control of physicians. But there are individual causes of behavior disturbances in children for which the physician can do something. I think it is fair to say—and psychiatrists are agreed on this—that if there is any chance of preventing the psychoneuroses of adults the job must be done during childhood. All one can do with the bulk of psychoneurotic adults—to quote Dr. A. A. Brill—is patchwork. Brill points out further that "the good one can do even with intensive treatment of adult psychoneurotics amounts to very little considering the enormous number of psychoneurotics and the very few doctors and the amount of time that it takes."

SUITABILITY OF THE PEDIATRICIAN AS CHILD PSYCHOLOGIST

I have taken as my theses in this paper that the subject of behavior disorders in children is one that the pediatrician, by training and experience, is eminently suited to handle; that the incorporation of psychologic care into the general program of child care requires no strange departure from traditional thought, and that the problems which arise require the same sort of analytic approach and offer the same sort of diagnostic challenge as do problems in the physical sphere.

Furthermore, the pediatrician alone is in a position to carry out a program aimed at promoting optimal psychologic health. He alone has the opportunity of recognizing difficulties early, before they have become firmly established. In private practice he needs no psychiatric social worker to ferret out the home and the idiosyncrasies of the parents. Generally, through years of association, he has come to know intimately the sort of environment from which his patient comes.

At present, in the matter of the behavior disorders, the pediatrician finds himself in a dilemma. On the one hand, he is assailed for his lack of interest in this subject, which, in the minds of some enthusiasts, transcends in importance all other aspects of child care; on the other hand, when he shows some interest in child psychiatry he is apt to be told that he is not equipped to delve into the depths of the child's mind and that he would do well to refer his patients' problems to those who are especially trained in this field.

Despite some reservations, pediatricians, by and large, are aware of their responsibilities for the psychologic care of the child. More than any other branch of medicine, pediatrics has been interested in the patient as a whole and has actively practiced prophylaxis and the promotion of optimal health for many years. But pediatricians, like practitioners in other fields, have been alienated from the handling of emotional problems by a point of view and a terminology which are unique in medicine. I refer to the custom among psychiatrists of speaking of schools of thought: the psychoanalytic school and its ramifications, the psychobiologic school, the behaviorist school, psychosomatic medicine and others. When one discusses nutrition, one is apt to mention the contributions of various men, like Alfred Hess, MacCollum and Elvehjem; in the field of allergy, there come to mind the names of von Pirquet, Schloss, Cooke and others. It would help to clarify matters greatly if psychiatrists were to do the same and were to speak of the contributions of Kraepelin, Freud and Gesell, instead of referring to schools of thought and practice, a custom which gives the impression that problems of the mind somehow require a point of view and an approach unlike those used in physical care.

A further reason why pediatricians have hesitated to incorporate psychologic care into their practice is that they feel that it requires the acquisition of a large fund of new information.

Here pediatricians are unduly modest. Actually, they have learned from their years of contact with patients a great deal about the mental and emotional development of children and about the attitudes of parents. They need, perhaps more than anything else, to acquire tags and labels for their knowledge; and they need a better integration of their information and a deeper insight into the types of behavior which come under their ken.

One of the objections to the handling of behavior problems by a pediatrician is that it requires more time than he can afford to give. This is only partly true. As one acquires facility in recognizing patterns of behavior and in assessing parental attitudes, the time necessary for each patient can be reduced. Furthermore, in an obscure problem of physical disease—for which the leads are too few or too many—history taking is time consuming, too.

At the risk of being dull and perhaps trite, I shall try to make this paper informative. My discussion falls under three headings: (1) the mechanisms underlying the behavior disorders, (2) the management of some of the common behavior disorders and (3) the promotion of optimal psychologic health.

MECHANISMS UNDERLYING THE BEHAVIOR PROBLEMS

Many of the disorders of behavior and feeling in children can be best understood as results of parental attempts at interfering with the developmental process. A knowledge of child development is just as valuable for psychologic care as it is for physical care. By development, or maturation, is meant the innumerable stages and processes through which the human form, functions and behavior pass from conception to maturity. Development is innately determined and can be influenced by environmental factors to only a minor degree.

A simple illustration of how the developmental process operates in the physical sphere is the case of height and weight. Growth during prenatal life is rapid, continuing apace until about the end of the first year. The tempo then slackens, and growth continues at a slow pace until about the fourth year, when a slight acceleration takes place, which lasts about two years. This is followed again by a period of slow growth until just before puberty, when there is another spurt which lasts about four years, after which slow growth continues until maturity is reached in the early twenties.

This is the shape of the growth curve for every normal child. The ages when the spurts take place vary in different children; the height

of the curve varies in different children, but the general shape of the curve is constant and cannot be altered except by drastic means.

Psychologic development has the same sort of regularity and the same inevitability. Motor skills, mental abilities, the capacity to respond emotionally to various situations—all these appear spontaneously as part of the developmental process. The extent to which the unfolding potentialities are used depends on the environment.

Take, for example, the application of the developmental concept to training to use the toilet. At some time toward the end of the first year or early in the second year, maturation of the proper innervation to the rectum and bladder takes place, making voluntary control possible. At about the same time, the cerebral cortex matures to the point at which there is some comprehension of the reason why one must wait until being put on the toilet. Readiness for training is an aspect of the developmental process. It appears spontaneously and inevitably. It is obviously impossible to train a child, in the proper sense, until such readiness is attained. Premature attempts at toilet training—just like premature attempts at training for other activities, for example, keeping a promise or learning to read—are unnecessarily difficult and doomed to failure. The child is unable to live up to expectations; the parent is annoyed and often exasperated, and the child senses the parental displeasure and is consequently unhappy. How the child will express his unhappiness will depend on his age, on his own personality make-up and on a multiplicity of other factors. In parental attempts at accelerating the developmental process, then, lies the mechanism for one group of behavior problems.

Failure to Meet Psychologic Needs of the Child.—The unfolding of the psychologic maturation sequence, like that of the physical, is obligatory and not optional. In order to attain optimal health, the maturing child must have an outlet for his developing skills and feelings, and it has now become customary to speak of the psychologic needs of the child, just as one speaks of the nutritional needs.

Otherwise stated, this means that when the child is ready to walk he must be permitted to walk; when he is ready to be trained to the toilet he must be given an opportunity to use the toilet, and when he is ready to dress and undress himself he should have an opportunity to do so. It means, too, that the child's need for affection, attention, praise and encouragement should be gratified.

Of prime importance for the proper emotional development of the child is gratification of the need for affection. This is a definite, organic requirement. From earliest infancy, the child must know that his mother loves him, and this love must be expressed to him in terms which he can comprehend. It is notable that mothering

and cuddling are universal among mammals, and they are widespread in lower forms, for example, among the barnyard fowl and the nesting birds. In the breast-feeding situation the baby gets an overt expression —for him—of maternal affection, and this is perhaps the greatest value of breast feeding. All along the line, as he grows up he needs constant reiteration of maternal affection.

Likewise, the greatest trauma which can be inflicted on the child results from lack of affection—that is, maternal indifference or rejection—and the severest behavior problems arise out of this situation.

Failure to supply an outlet for the child's emerging potentialities and to gratify his emotional needs then constitutes a further mechanism leading to behavior problems.

Attempts to Retard the Developmental Process.—Another group of problems arises when attempts are made to retard the developmental process. The growing-up process implies not only the acquisition of new modes of behavior but the relinquishing of outgrown patterns as well. There is an analogy here to nutrition; a diet entirely of milk, which is adequate for an infant, is harmful if continued during childhood.

A simple example of psychologic retardation may be seen in the eating situation. Despite the fact that a child is ready, the mother often hesitates to make the transfer from mushy to lumpy foods and from bottle to cup, because she fears he will not eat as much as he did formerly. Frequently she does not allow him to eat by himself, either because he may not eat as much or because the process is too messy. Many mothers continue to dress their children and bathe them after they are ready to do these things for themselves. Later, on another plane, the mothers undertake to fight their children's battles for them.

Thus in parental attempts at retarding the developmental process, there is another mechanism causing problems of behavior and feeling.

Etiologic Factors in Behavior Disorders.—The developmental viewpoint is a relatively new one, and pediatricians are indebted largely to the Yale pediatrician, Dr. Arnold Gesell, and his able group of co-workers for ideation and data in this field, The viewpoint is in sharp contrast to that of the behaviorists, whose ideas were dominant in child rearing until rather recently. The behaviorists largely disregarded the emotional needs of the child, and they were, to a considerable extent, responsible for the rigid attitude of pediatricians of the past.

The change in attitude toward child rearing which has taken place in recent years is a wholesome one. That children from earliest infancy are individuals with emotional and mental as well as physical attributes is a concept which is self evident and which has come to stay.

However, a note of caution is in order. Whereas children were formerly subjected too much discipline, many now have too little. The reins, once held too tightly, have been slackened to the point at which many children lack direction entirely. To allow children always to have their way is as bad as—perhaps worse than—not to allow them enough freedom. Implied in the permissive attitude recommended by many, is the assumption that children intuitively select the optimal course. This is true to only a limited extent. Even in the studies on self feeding, the battery of foods presented to the children were necessarily preselected. Children need direction, and they need discipline which defines the limits of their freedom.

In general, the etiologic factors leading to behavior disorders are multiple. This is true also of the causes of physical ailments, although here the pediatrician can often work on one factor in the complex of causality (as in the case of sulfonamide therapy for various infections) and effect a cure. In the management of the behavior disorders, however, it is generally necessary to consider several factors.

For convenience, the etiologic factors leading to behavior disorders may be divided into two groups: those contributed by the individual and those contributed by the environment.

Factors Contributed by the Individual: The innate emotional endowment. It is reasonable to assume that, just as children vary in all their physical attributes, just so do they vary in their capacity to respond emotionally. One child will react with fear to a given situation which will have no effect whatsoever on another. It is a mistake to attribute this difference in response entirely to conditioning. In the same way children differ in their innate capacity to react with anger, aggressiveness and self dependence. It is conceivable, too, that in some children the emotional balance is sturdy and difficult to upset, while in others it is delicate and readily disturbed.

Not long ago, I saw a little girl of 8 who was actively, overtly rejected by her mother. The mother started one interview by saying "Doctor, you must do something to this child. If she continues to misbehave, I am afraid that I shall do her some harm." From what is known of the effects of parental rejection on child behavior, one should have expected a badly traumatized child. Actually there was little wrong with the child. She was an active, healthy, happy, robust youngster. I believe the mother's principal source of annoyance was that the child was so little disturbed by her mother's aversion to her.

The innate intellectual endowment. One of the most important factors to be considered in analyzing the mechanism of a behavior problem is the innate intellectual endowment of the child. It is apparent that the intelligence will be closely related to difficulty at school.

That a child with poor mental endowment will do poorly at school is to be expected. But a child with superior intelligence sometimes has difficulty as well. He may be bored with the simplicity of the school work and with the plodding efforts of his less brilliant classmates. He may not listen to what is going on in the classroom, and hence, when he is called on, he cannot answer properly. He may misbehave at school and in this way invite the antipathy of the teacher. I know one boy with an intelligence quotient of over 160 who takes it on himself to correct the school teacher's erroneous statements. This boy, bright but unbridled, should be advanced to a higher grade (this does not always correct the situation), but it is naturally hard to convince his teacher that the boy should be rewarded for his impertinence by being promoted.

Emotional disturbances are often related to the level of intelligence. Many parents fail to recognize the mental inadequacies of their offspring, and, out of ignorance or false pride, they attribute the difficulties at school to laziness and lack of interest. Conflicts arise; there are arguments, scoldings and spankings, and these further interfere with the child's school performance.

One can do much for the moron by proper training and avoidance of emotional problems. Studies have shown that there are plenty of jobs which he can hold down satisfactorily.[3] Pitfalls which confront the moron during childhood are overprotection, on the one hand, and undue pressure to compete with normal children, on the other. From earliest childhood, the moron must be taught to carry his responsibilities within the limits of his mental capabilities, and he must not be expected to compete on an equal level with children of normal mentality.

The child with superior mentality also has his emotional troubles. They are the difficulties which one would expect in, for example, a 10 year old child with a 14 year old mentality. Such a child is generally in a grade with bigger children. Consequently he is unable to compete adequately in sports. Furthermore, his classmates are adolescents while he is still emotionally a child.

Variations in intelligence, then, may serve as the focal point around which a behavior problem begins.

The innate motor endowment. Here, too, there is great individual variation. As is well known, some children and adults show great natural aptitude at such activities as dancing and sports while others are clumsy and awkward. In many instances no explanation can be found, but in some the awkwardness is on the basis of conversion from left-handedness to right-handedness.

3. Louttit, C. M.: Clinical Psychology: A Handbook of Children's Behavior Problems, New York, Harper & Brothers, 1936, p. 151.

During the past year I saw a 13 year old boy about whom one of the principal complaints was motor awkwardness. That an adolescent should be awkward is not unusual. But in this boy, the difficulty had been first observed at the age of 3 years and had persisted throughout childhood. Though the boy was popular among his classmates and well grown, he was always the last to be chosen when teams were made up. He had a superior intelligence quotient. Early in childhood the boy had been ambidextrous, but he had been trained to use the right hand. There was no language difficulty aside from poor handwriting. Tests showed that he was left eyed, probably left-handed and right footed. The tests for handedness did not give clearcut results, and there seemed to be considerable confusion on the part of the boy as to which hand he was to use.

Sex. The sex of the child has some bearing on the character of a behavior disorder. Girls are more likely than boys to show fear reactions, especially anxiety states. Aggressive reactions are more common in boys than in girls.

Race. Another factor is race. Bender [4] has pointed out specific reaction patterns commonly met with in disturbed Negro children—a certain passivity, blocking and mutism—a reaction almost amounting to catalepsy. She did not venture to say whether these are innately determined or whether they are cultural. Negro children also suffer because of their racial unpopularity, and a similar situation exists in the case of children whose nationality or religion differs from that of the majority group.

Constitutional defects. There are several groups of disorders the origins of which lie in what may be loosely termed constitutional defects. According to Orton,[5] Cobb [6] and others, difficulties in the language functions—speech, reading and writing—fall into this group. Although stuttering may be solely psychogenic in origin, it is likely that there is usually a structural abnormality in the brain. Persons with such an abnormality in the language centers, when exposed to emotional stress, are much more likely to acquire speech defects than are structurally normal persons—and the same is probably true of the other language functions. There is reason to believe that the language difficulties are related to alterations in lateral dominance—handedness, eyedness and footedness. The language difficulties are much more common in boys than in girls and are generally hereditary.

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4. Bender, L.: Behavior Problems in Negro Children, Psychiatry **2**:213 (May) 1939.

5. Orton, S. T.: Reading, Writing and Speech Problems in Children: A Presentation of Certain Types of Disorders in the Development of the Language Faculty, New York, W. W. Norton & Company, Inc., 1937.

6. Cobb, S., and Cole, E. M.: Stuttering, Physiol. Rev. **19**:49 (Jan.) 1939.

There is reason to believe that enuresis, too, generally has its origin in a constitutional defect,[7] in this case in the innervation of the bladder. In most instances enuresis is hereditary; it continues from infancy, and it is associated with frequency and urgency and often with day wetting. The capacity of the bladder is often small as measured by bladder-filling studies, and, though the enuresis generally ceases in adult life (except under conditions of stress, such as war), frequency and urgency may persist. Here, as in the case of the language functions, emotional stress plays a prominent, but secondary, role.

In this group, also, may be included certain types of vomiting. There are some persons who can vomit easily, and there are others to whom it is a major episode.

Gross disease of the brain. Electroencephalographic studies reveal that in a not inconsiderable number of children gross structural changes in the brain are associated with disorders of behavior and feeling. Prominent among these disorders is hyperactivity—not the sort which is frequently seen in children but the severe kind in which there is ceaseless activity, tearing of clothes and destructive tendencies. The exact nature of the structural changes in the brain varies; there may be old encephalitis, injuries at birth or congenital defects.

Physical defects. Physical defects in children may lead to behavior disorders. Crippled children are exposed to many psychic hazards. Deluged by sympathy and overserved by their parents, they are apt to grow up as demanding, complaining persons, feeling that society owes them a perpetual debt. Much can be done to avoid these undesirable attitudes and to build for happy adulthood. A crippled child should have duties and responsibilities within the limits of his deformity, and he should be held to these just as strictly as is a normal child. In addition, he needs to have a constructive attitude toward life, which will lead him to accept his deformity and compensate for it.

There are other ways in which physical defects may lead to behavior problems. A visual or aural defect may lead to school difficulty and a train of auxiliary problems. A child with strabismus may be teased and thus refuse to go to school. Fatigue or chronic illness may lead to hyperirritability.

The individual factors, then, to be considered in analyzing a behavior disorder are attempts at interference with the unfolding of the developmental patterning in the direction of either acceleration or retardation; failure to gratify the psychologic needs and overgratification; the individual's innate emotional, mental and motor endowment; sex and race or nationality of the patient; constitutional defects; gross structural changes in the brain, and physical defects.

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7. Bakwin, H.: Enuresis in Children, J. Pediat. **12**:757 (June) 1938.

Factors Contributed by the Environment: The principal environmental factors influencing psychologic development during childhood are four: the parents, the siblings, the school and the playmates. Of these, by far the most important is the attitude of the parents, particularly the mother.

The parents. During recent years a terminology has been developed to characterize the parental attitudes. This terminology is simple and useful, and one will find it worth while to adopt it if one has not already done so. One speaks of parents as overaffectionate, underaffectionate, rejecting or merely indifferent. There is overanxiety, overprotection, overindulgence, perfectionism and overauthority. Not only do these terms offer a shorthand method for expressing parental attitudes; they "point up" the qualities in parents which are important in influencing child development. It is worth noting that some of these, like overanxiety and overaffection, express emotional states while others, like overprotection and overindulgence, indicate modes of behavior.

In most instances it is a relatively simple matter to assess the parental attitudes from observation. There is one parental attitude, however, which is often concealed from the physician and which may be the origin of the severest behavior problems. I refer to parental rejection. Parents appreciate that this attitude is undesirable, and they try their best to hide it—sometimes even from themselves. Whenever a really severe behavior problem presents itself, parental indifference or rejection should be suspected. It is my impression that indifference is more highly traumatic for the child than actual rejection. It is the emotionally indifferent atmosphere of the hospital which makes residence there for infants so highly lethal. The child exposed to this sort of environment is, as it were, up against a blank wall.

Perhaps the most frequent harmful attitude met by a pediatrician is parental overanxiety. In good part, this is a reflection of the general state of mind to which I referred earlier. But there are other reasons. Many mothers have the false notion that child rearing is a science, that there is one correct way of bringing up a child and that all other ways are wrong. Of course, the pediatrician knows the correct way, she believes. I am afraid that pediatricians are in no small way responsible for this parental misconception. It was not long ago that they themselves thought that they knew just how much a baby ought to eat and when; just how long he ought to sleep, and just when he ought to be trained in the use of the toilet. In general, the first task of a pediatrician, when he sees a newborn infant, is to try to allay maternal anxiety.

The parental attitudes may have their origin in ignorance of proper methods of child rearing, or they may stem from deep-seated personality

distortions. As one might expect, if the latter is the case the disturbance in the child is likely to be severer and therapy less successful. Neurotic adults frequently marry not out of real affection but rather to get out of an environment—that is, their home—which they find distasteful or even hateful. Marriage on this basis is not likely to be a happy affair. It is accepted only tentatively. The first child is often rejected because he is looked on as a bond sealing an undesirable marriage more closely. Sometimes the reverse happens, more often with the second child, and the neurotic mother pours out all her pentup affection on the child.

A typical example of the rejecting mother is the one I mentioned earlier who said that she was afraid that she would harm her 8 year old girl. This woman had many complaints about the child: The child was disobedient; she hit her little brother; she showed no affection for her mother, and she showed an abnormal interest in men.

The mother's family history was interesting. She was the oldest of three children. Her father was a domineering person, whom she heartily detested. Her own mother was a weak, sickly, helpless creature, and from an early age the complaining mother had managed the household. Her sister was 11 years younger than she and was pretty. My patient's mother said that she was the ugly duckling.

She married a small shopkeeper when she was in her early twenties. I never met him, but I understand that he was a colorless person who took little interest in the family and who spent his days and evenings at his business.

The first child, the patient, was born five years after the parents were married, and a second child, a boy, was born four years later. At first the mother assisted her husband in his business, but shortly after the birth of the second child she opened a store of her own at the management of which she worked hard and was successful.

She became suspicious of her 8 year old daughter's interest in men after a trip to Florida. Here the child had met a middle-aged man who seemed to like her and gave her little gifts, such as candy and toys. On her return to New York the little girl often spoke of the man and even wrote him a letter. All this seemed extremely suspicious to the mother. She was also annoyed that the child often sat before the mirror and admired herself.

It requires no deep psychiatric insight to analyze the personality attributes of this mother and their origins. Her home before marriage had been an unhappy one, what with the bullying father and the weak, self-centered mother. She had been made the drudge. Naturally she was jealous of her younger sister, who did not have to carry the burden of the household. Moreover, the younger sister was pretty, and she was not.

Marriage for this woman was certainly not a love match but a means of getting out of her unhappy home. Though she rejected her children, particularly the little girl, she was a conscientious mother and gave them every attention. She could not understand why the little girl did not love her, and she did not realize that she was transferring to her daughter the jealousy and resentment which she felt toward her sister. Her youthful training as a manager of her parents' home had made her self reliant, and she was determined to get some personal satisfaction out of life—hence, her venture as a shopkeeper.

There is no need to pursue this case further. I present it as an example of a parental attitude based on a deep-seated personality defect rather than on parental ignorance.

A point worth keeping in mind—and one about which there is little mention in the literature—is that the parental attitudes are not necessarily fixed. Changes frequently take place. A young couple may not have wanted a child, and they may at first be indifferent to it. But if they are normal—and perhaps are encouraged by the pediatrician —they may come really to love their child.

A change of attitude is sometimes seen toward the overindulged child. The child at first is showered with affection and attention; but later, when he starts to act up and misbehave, perhaps after the birth of another child, the parents may actually come to reject him.

Such factors as the paternal intelligence, educational status and physical health may also influence the development of behavior disorders. Parental incompatibility is usually extremely disturbing to children. The socioeconomic status of the family is generally not an important factor to young children, but it may be as children approach adolescence. Differences of opinion between the parents regarding discipline is a frequent and potent cause of difficulties.

In some instances, imitation of parental behavior is the basis for a problem. The parents of a girl of 15 complained, among other things, that she was argumentative and heated in her discussions. It happened that I had known the father of the patient since he was a youngster. She could not have been worse in this respect than her father. I may say that the parents had failed entirely to appreciate that the girl was patterning her behavior after her father's.

Religious differences between the parents sometimes lead to behavior problems in children. A few weeks ago I saw a little girl because she had attacks of vomiting. The history as given by the mother was a classic one for the condition known as school vomiting. The child vomited each morning on the way to school, sometimes before leaving home. On week ends she was all right. She attended a Catholic parochial school and, according to her mother, was frightened by the nuns. It seemed like a straightforward story; the child found school

disagreeable, hence vomited. But it turned out that there were complicating factors. The father was Catholic, the mother Baptist. Apparently the mother, who was the dominant member of the family, was acquiescing in an earlier promise to bring up the child in the Catholic faith, but she was not reconciled to this idea and sought a way out. She had subtly suggested to her little daughter fear of the nuns as a device for getting her into a public school.

Occasionally the grandparents or the nurse play a determining role in the genesis of behavior problems.

The siblings. Siblings constitute part of the normal home environment of the child. Where the parental attitudes are proper, there are ordinarily no serious difficulties among the children. For children to be jealous of one another is natural; for a first child to "act up" a little after the birth of a second child is usual and to be expected. But sibling rivalry is a real problem only when there is a background of trouble stemming from the parental attitudes.

A common reaction in children of 3 or 4 years of age following the birth of a younger sibling is what may be termed an infantile reversion. The older child wants to suck from a bottle and wear diapers—and he often wets and soils himself while sitting on the mother's lap. This behavior is of no great significance and disappears spontaneously.

The school. The school is the child's workshop, and most children take it very seriously. It was found at Bellevue Hospital—and this has been the experience elsewhere—that the chronically ill children were much better contented when school work was provided than they had been before. Problems which arise are difficulties in adjusting to the school routine and the other children and difficulties with school work.

Playmates. Playmates constitute an integral part of the child's life. Children do not play together until they are about 4 years old. Before this they enjoy playing where other children are present, but they do not play together to any great extent.

Lasting friendships are not ordinarily formed until the child is in high school. As is to be expected, problems arising from association with playmates tend to increase as the child grows older and the links to the home grow weaker. An important factor to keep in mind in this relation is the child's desire to conform to the mores of the group. This is especially strong in the adolescent, but it is seen even in the young child. I know of a little boy who steadfastly refused to stay at nursery school. It took some time before his mother discovered that the reason was that all the other children wore overalls while her son did not.

Problems arising from the influence of playmates are most commonly seen during adolescence.

MANAGEMENT OF COMMON BEHAVIOR PROBLEMS

Before discussing some of the commoner behavior problems of children, it may be well to ask: What is a behavior problem? Does the child who wanders away from home because he is afraid to face his parents with a bad report card and take a whipping present a behavior problem? Is for that matter the boy who runs away from home to seek wider horizons a problem child? This was a not uncommon mode of behavior in America only a few years ago. Many men of eminence made their beginnings in this way. Is the boy who as a member of the gang snatches fruit from the pushcart peddler a delinquent? Or is it more likely that the boy who remains apart and refuses to go along with the gang is the problem child? Is the child a problem because he refuses to eat what he does not like simply because the parents (probably erroneously) believe it is good for him?

There is reason to believe that thumb sucking in infants and young children is not a behavior problem or, for that matter, an undesirable habit. It probably represents the same sort of a behavior as cigaret smoking, gum chewing and munching in adults. Many so-called problems arise as a result of the parents' preconceived ideas as to what sort of person their child is to be. A complaint voiced by many parents is that their child is not aggressive enough. It is common for fathers who spend their days in white collar occupations to want their children to be pugilists. What can these parents expect, considering the example they have set?

The first thing to decide, then, in approaching a behavior problem is whether there really is a problem or whether one is dealing with a variant of normal behavior.

Of the behavior disorders seen in pediatric practice, anorexia still remains the most frequent. The principal etiologic factors generally are parental overanxiety for the welfare of the child, parental ignorance of the principles of nutrition, and improper training in eating technics.

It has been my experience that anorexia can be successfully treated in the large majority of instances. First of all, the parents must be reassured that the condition need not concern them greatly, that the physical status of the child is normal and that the child's intake of food is adequate. The decreased need for food as the growth rate declines should be pointed out.

The parents should know that a discriminating attitude toward food on the part of the child is part of the growing-up process and that the child's discriminating tastes should be respected. They should know that children vary in their gaining of weight just as they do in all other attributes.

The child should have a free choice of foods. There should be no insistence on this or that food. Food should be prepared attractively and temptingly. The mother should equip herself with a good cookbook and forget calories and vitamins. Young children have definite preferences. They like meats, butter with or without bread, fruits and often milk. What better foods are there? Cereal is the food most widely disliked, and it can be safely omitted from the diet.

The question of entertainment at mealtime requires some discrimination. It is certainly overdone by most parents. Yet to adults eating is a social function, and it is to children too.

Training for self feeding is an important part of the treatment.

In occasional instances, anorexia is an expression of the child's unhappiness. In these circumstances it is, of course, necessary to investigate the cause of the child's emotional discomfort.

Though, as already stated, I do not believe that thumb sucking is a problem which requires treatment in young children, the parents need some explanation about it. They should be reassured that it is innocuous, that it will not deform the teeth or palate, that it is not related to masturbation and that attempts at correction may lead to difficulties. When a child past the age of 2 continues to suck the thumb during the day, it means that he is bored, unhappy or overfatigued and the reasons for these feelings then require investigation.

Disciplinary problems are common. Discipline is necessary for the child. It does not necessarily imply punishment, although, when indicated, punishment should be administered. Most disciplinary problems arise out of differences of opinion on the subject between parents. One parent says one thing, the other says something else and the child does as he pleases. The parents should be told that there is no single right way for the child to behave. The most important thing is that the parents should agree. Disciplinary situations are usually difficult to handle, since the parental differences of opinion are often expressions of the parents' own personality difficulties—or of incompatibility.

A common and interesting group of problems is that relating to the language functions. I shall discuss only one, specific reading disability, since, though easy to detect and correct, it is often missed and may lead to much misery. The child with this difficulty is unable to read as well as might be expected from his general intelligence. The defect may occur in children of all levels of intelligence, high as well as low. It is much commoner in boys than in girls.

There is reason to believe that the basis for this condition is generally a structural defect in the language centers and that it is related to alterations from the normal in lateral dominance. In some instances there are gross defects of vision. In others there are less obvious visual

defects, such as poor fusion. In normal readers the eyes move smoothly from left to right, but in poor readers the eyes fix on words and move back and forth irregularly.

An emotional overlay intensifies the difficulty. Children with reading disability approach reading tense and fearful, and this is likely to worsen their performance.

It is important that this defect be recognized early, since much can be done by proper training. It tends to correct itself by 12 years or so, but much harm can be done before this if the disability is neglected. School teachers should recognize this condition, and they often do; but it is often missed by them, and the pediatrician will do well to look for it.

THE PROMOTION OF OPTIMAL PSYCHOLOGIC HEALTH

Desirable as it is for the pediatrician to have an understanding of the mechanisms underlying behavior disorders and to grasp the principles of therapy, it is even more important that he apply himself to the promotion of optimal psychologic health just as he does to the promotion of physical health. Here the pediatrician has a unique opportunity.

The program of psychologic care[8] should include, first of all, an attempt to instil proper parental attitudes. The pediatrician can do much to allay parental overanxiety. Often he can do something to influence overaffection and underaffection—at any rate he should try. He can use his influence to combat overprotection, overindulgence, overauthority and some of the other undesirable parental attitudes.

He can give valuable advice on proper methods of training and discipline, and he can point out the hazards of improper training.

The pediatrician must acquaint himself with available data on the mental, emotional and motor development of the child, and he must educate his patient along these lines.

COMMENT

I am not one of those who consider child psychology and psychiatry the most important parts of pediatrics. To pediatricians interested in the child as a whole, no one aspect is more important than another. I do feel, however, that they have neglected these branches of the specialty too long. They have passed on their obligations in this field to nonpediatric groups, who, with the best of intentions and with a great deal of first-rate work to their credit, nevertheless lack the breadth of viewpoint and the objectivity of the pediatrician.

8. Bakwin, R. M., and Bakwin, H.: Psychologic Care During Infancy and Childhood, New York, D. Appleton-Century Company, Inc., 1942.

I look forward to the time when in caring for a child, whether sick or well, whether in the home or the hospital, in public clinic or in private office, the same attention will be paid to the mental, emotional and motor attributes as is now given to the physical. I look forward to the time when disorders of behavior and feeling will be attacked by the pediatrician with the same vigor, courage and ingenuity that have been successful in the elimination of so many of the physical disorders.

I do not anticipate the amazing success in preventing the psychoneuroses which has been so rewarding in the case of the physical ailments, but I do see a widening horizon which should enrich pediatric investigation and practice for many years to come.

SUMMARY AND CONCLUSIONS

1. Pediatrics has been eminently successful in reducing morbidity and mortality from physical causes among children.

2. Pediatrics has done little or nothing to prevent the development of psychoneuroses during childhood and later life.

3. The task of promoting optimal psychologic health and of preventing psychoneuroses in adults is the pediatrician's. For this task he is well suited by training and experience.

4. The principal mechanisms underlying the behavior disorders of the child are outlined.

5. Certain common behavior disorders in the child are discussed.

6. A program for promoting optimal psychologic health is outlined.

7. Interest in the psychologic aspects of the child will enrich pediatric investigation and practice.

132 East Seventy-First Street.

OUTLINES FOR WELL BABY CLINICS

I. Recording Development of the First Twelve Months

C. ANDERSON ALDRICH, M.D.
Director, Rochester Child Health Project; Section on Pediatrics, Mayo Clinic
AND
EDITH S. HEWITT, M.D.
First Assistant, Section on Pediatrics, Mayo Clinic
ROCHESTER, MINN.

DURING the past thirty years infant welfare clinics have developed from mere feeding stations, where nurses or doctors or both prescribed formulas and diets for underprivileged patients, to the modern well baby clinics. Therein an attempt is made to supervise not only diets but also other matters, such as formation of basic habits and social adjustment and their relation to normal growth and development. This is in line with modern concepts of treating the whole baby rather than individual parts of him, such as the throat or gastrointestinal tract, or specific diseases with which he may be afflicted. In spite of the tendency of the clinics to develop as has been described, the records of babies in most clinics rarely give adequate descriptions of growth and development, formation of habits or social adjustment. An over-all picture of a baby is seldom available.

When a young physician without previous experience in the work of an infant welfare clinic is suddenly confronted in such an establishment with many mothers and their babies, he finds himself uninformed as to how to question the mothers, how to observe the babies or how to record the information he obtains. If the information obtained at each conference can be systematically recorded, a comprehensive picture of each child can be developed. Later, the many pictures thus obtained can be reviewed to increase general knowledge of the processes of growth and development.

To accomplish the two purposes of (1) teaching a young physician his work and (2) obtaining descriptions of the growth and development of individual babies, the Rochester Child Health Project has made up a set of cards from which the inexperienced physician can ascertain the items to be considered at each conference. These cards are reminders only; they are not record cards. A separate card is provided for each of the 12 months of the first year of life. A thumb index cut into the

From the Rochester Child Health Project; Section on Pediatrics, Mayo Clinic.

cards makes for easy selection of the card which corresponds with the month under consideration. Each card is perforated in a lower corner, and the set of twelve cards is held together by means of a paper fastener so that each card can be made visible easily.

On the cards are notations under each of the following headings: (1) development, (2) schedule for the baby's day, (3) food, (4) social adjustment and formation of habits and (5) physical state. In this paper the material on each card is listed, and any necessary explanation of notations on the card is given in the accompanying explanatory notes.

CARD 1. SUBJECTS TO BE CONSIDERED AT AGE OF 4 TO 6 WEEKS

1. *Development.*—Smiling and vocalizing may appear. Explain future development of voluntary acts and cephalocaudal law.

2. *Schedule for the Baby's Day.*—Discuss feeding schedule and baby's needs as manifested by crying. Omit 2 a. m. feeding if baby sleeps through night.

3. *Food.*—Adjust formula or breast-feeding technic. Prescribe cod liver oil and orange juice.

4. *Social Adjustment and Formation of Habits.*—It is too early for training in elimination. Child sleeps most of the time.

5. *Physical State.*—Colic may require treatment.

Explanatory Notes Relative to Card 1.—The first voluntary act of a baby is smiling, and this develops into a definite response to the mother at about the age of 1 month. Smiling is soon accompanied with "oo" and "ah" sounds. If these accomplishments have not yet emerged when the baby is first brought to the clinic, tell the mother to watch for their early appearance.

According to the so-called cephalocaudal law, which applies to all animals, the maximal growth impulse begins at the head end and passes slowly toward the tail. In similar sequence, the nerve fibers grow out from the nerve cells of the brain to the different muscle groups. Thus, the baby performs intentional acts first with the muscles of the face, next with the muscles of the throat and later with the muscles of the arms, trunk and legs.

Babies demonstrate their need for food by crying. If the baby is fed in sufficient quantity when he manifests his need for food, crying usually is easily controlled. Within the first month crying for food usually comes at regular intervals, so that, if allowed to do so, the baby will develop his own feeding schedule. Occasionally adjustment of feeding may not be sufficient to control the crying, and relaxing and sedative drugs may be required.

CARD 2. SUBJECTS TO BE CONSIDERED AT AGE OF 2 MONTHS

1. *Development.*—Smiling and vocalizing well developed. Begin to watch for head control and for hand to mouth tendency.

2. *Schedule for the Baby's Day.*—Crying should be less of a problem. Feeding rhythm is usually established. The 2 a. m. feeding usually has been omitted.

3. *Food.*—Prescribe formula if needed.

4. *Social Adjustment and Formation of Habits.*—Sleeping habits have become regular. Bowel movements have increased in regularity.

5. *Physical State.*—Has any idiosyncrasy to food developed? Signs are rash, vomiting or colic.

Explanatory Notes Relative to Card 2.—By "head control" is meant the ability of the baby to hold the head firmly in the axis of the inclined torso. The test is: Holding the baby by the arms, pull him to the sitting position from the dorsal recumbent position and lower him again to that position. If the head falls back, head control has not been established. Usually this type of control is acquired in the third month of life.

As the baby becomes able to move his hands about and put them together, he will also be likely to put his fingers or thumb into his mouth; thus, thumbsucking is part of normal behavior in the first few months, although it is still involuntary.

CARD 3. SUBJECTS TO BE CONSIDERED AT AGE OF 3 MONTHS

1. *Development.*—Head control attained. Watch hands for possible appearance of next sign of development (explained on card 4).

2. *Schedule for the Baby's Day.*—Compulsive crying about gone. The 2 a. m. feeding definitely dropped.

3. *Food.*—Prescribe formula if needed. Gradually introduce cereal if baby is advanced or very hungry. Egg yolk may be added.

4. *Social Adjustment and Formation of Habits.*—Spoon feeding may be started if baby can carry solid food from front of mouth to pharynx.

5. *Physical State.*—Record anything noteworthy.

Explanatory Notes Relative to Card 3.—Compulsive crying, or crying without demonstrable reason, which may have required the use of drugs for control, usually disappears at this age.

Also at this age, neuromuscular development of the tongue and throat usually has progressed so that solid food placed on the baby's tongue can be passed back to the pharynx in a swallowing motion. If such is the case, he is ready to start to eat cereal from a spoon. If he has not yet reached this stage of development, he will push out of his mouth any solid food put onto his tongue.

CARD 4. SUBJECTS TO BE CONSIDERED AT AGE OF 4 MONTHS

1. *Development.*—Watch baby play (work) with his hands. He reaches out for objects and attempts to roll over. Baby may begin to help hold bottle with hands.

2. *Schedule for the Baby's Day.*—The 10 p. m. feeding possibly omitted.

3. *Food.*—Most babies receive cereal by spoon.

4. *Social Adjustment and Formation of Habits.*—Baby starts to laugh. Watch for regular recurrence of bowel movement.

5. *Physical State.*—Record anything noteworthy.

Explanatory Notes Relative to Card 4.—It is interesting and amusing to watch the baby look at his hands, put them together, pull them apart and begin to reach for objects. It seems to be play to the observer, but it is work for the baby. He is developing a new skill and practices it most of his waking time.

CARD 5. SUBJECTS TO BE CONSIDERED AT AGE OF 5 MONTHS

1. *Development.*—Control of hands well established. Baby may roll over from back to abdomen. He sits like a frog and cranes his neck forward.

2. *Schedule for the Baby's Day.*—Ten p. m. feeding usually is omitted.

3. *Food.*—Strained vegetables are introduced, one at a time.

4. *Social Adjustment and Formation of Habits.*—Baby can't be left alone on the bed because he rolls about.

5. *Physical State.*—Record anything noteworthy.

Explanatory Notes Relative to Card 5.—When strained vegetables are introduced, if the same one is given several days in succession the mother can note whether it causes any symptoms which indicate that it does not agree with the baby, such as vomiting, diarrhea or cutaneous rash. When the mother is sure of the way the baby reacts to the first vegetable, she can stop the use of that one and try a second one in the same manner. After the trial period, she can continue daily to feed the baby those vegetables that do agree with him.

CARD 6. SUBJECTS TO BE CONSIDERED AT AGE OF 6 MONTHS

1. *Development.*—Baby rolls over, sits better and may sit up alone.

2. *Schedule for the Baby's Day.*—Ordinarily there is no change.

3. *Food.*—Substitute vegetable soup at 6 p. m. for second cereal feeding.

4. *Social Adjustment and Formation of Habits.*—If baby sits up, "catching" of bowel movement may be started.

5. *Physical State.*—Record new teeth as they erupt from now on.

Explanatory Notes Relative to Card 6.—When neuromuscular development has advanced so that the baby can sit well alone, bowel training ("catching") can be started. By observation, the mother can tell when the baby feels the mass movement of the intestines; that is, when the contents are pressed down into the rectum and set up the impulse to defecation. That is the moment for her to place the child on the nursery chair or toilet seat. At first it is the mother who is trained, and success depends on her ability to "catch" the stool. If stools come at irregular intervals, the mother should not attempt this.

CARD 7. SUBJECTS TO BE CONSIDERED AT AGE OF 7 MONTHS

1. *Development.*—Usually baby sits without support. He may crawl or otherwise move short distances.

2. *Schedule for the Baby's Day.*—If baby sleeps late in the morning, he may have cereal and milk when he awakens and milk only at 10 a. m.

3. *Food.*—Potato and cooked fruits may be added. If baby can chew, give coarser foods.

4. *Social Adjustment and Formation of Habits.*—Watch for chance to "catch" bowel movement. Watch for consonants in vocalizing. Watch for imitation.

5. *Physical State.*—Start immunizations.

Explanatory Notes Relative to Card 7.—Soon after the baby can sit alone readily, he is likely to start to move about. There are many ways in which babies do this: Some sit up and push themselves around with their feet and legs; others lie on the abdomen and push with both arms and legs; others lie on the back and move by arching up onto heels and head, and still others may roll over and over. If, in any manner, the baby can reach objects which are a foot or so away from him, he has definitely started to move about.

CARD 8. SUBJECTS TO BE CONSIDERED AT AGE OF 8 MONTHS

1. *Development.*—Usually baby gets about on floor. A play pen is useful. Watch for prehension. He pulls himself to standing position. Still watch for chewing motions; if present, give chopped or mashed food.

2. *Schedule for the Baby's Day.*—May have early cereal feeding and milk only at 10 a. m.

3. *Food.*—Scraped beef or liver.

4. *Social Adjustment and Formation of Habits.*—Continue attempts to "catch" bowel movement. Do not train for urination. Continue watching for consonants in vocalizing and for imitation.

5. *Physical State.*—Continue immunizations.

Explanatory Notes Relative to Card 8.—By prehension is meant the ability to pick up small objects with the thumb and forefinger. Previous to this stage of development, objects are picked up by using all the fingers together, in a "raking" motion.

CARD 9. SUBJECTS TO BE CONSIDERED AT AGE OF 9 MONTHS

1. *Development.*—Creeping, prehension, pulling to standing position and chewing motions usually have been manifested. Put empty cup on tray for practice.

2. *Schedule for the Baby's Day.*—Some need only three meals.

3. *Food.*—Add desserts, zwieback and crackers.

4. *Social Adjustment and Formation of Habits.*—Continue "catching" of bowel movements. Do not train for urination. Continue observation of talking, use of consonants and imitation.

5. *Physical State.*—Continue immunizations.

Explanatory Notes Relative to Card 9.—All items are self explanatory.

CARD 10. SUBJECTS TO BE CONSIDERED AT AGE OF 10 MONTHS

1. *Development.*—Baby can stand and may walk with help. He can chew. Put spoon and empty cup on tray for practice.

2. *Schedule for the Baby's Day.*—May need only three meals.

3. *Food.*—Add whole egg and cottage cheese.

4. *Social Adjustment and Formation of Habits.*—Continue "catching" of bowel movements. What words can the baby say? How does he play?

5. *Physical State.*—Continue with immunizations. Is fontanel closed?

Explanatory Notes Relative to Card 10.—All items are self explanatory.

CARD 11. SUBJECTS TO BE CONSIDERED AT AGE OF 11 MONTHS

1. *Development.*—Baby walks better with help and may stand alone.

2. *Schedule for the Baby's Day.*—Probably needs only three meals and may eliminate morning nap.

3. *Food.*—Give bacon and lumpy foods.

4. *Social Adjustment and Formation of Habits.*—Continue attempts to "catch" bowel movement. Baby repeats syllables ("dada").

5. *Physical State.*—Continue immunizations. Fontanel may be closed.

Explanatory Notes Relative to Card 11.—Probably by now the baby goes to sleep soon after his evening meal and sleeps until about time for the family beakfast. If so, he has automatically placed himself in readiness for three meals, provided he stays awake all morning. However, some children may wish to continue the morning nap with a feeding before it or to have light refreshment in the afternoon. There is no reason why bottle feeding should be stopped abruptly. If the baby is allowed to continue, especially with the bedtime bottle, for several months, he is less likely to start the thumbsucking habit.

With the addition to the diet of foods containing more roughage, there is liable to be a tendency to frequency of bowel movement and difficulty in "catching" them all. The mother should be warned that this is a physiologic reaction and not a relapse in bowel control.

CARD 12. SUBJECTS TO BE CONSIDERED AT AGE OF 12 MONTHS

1. *Development.*—Baby may stand and possibly walk alone.

2. *Schedule for the Baby's Day.*—Usually takes only three meals. May be able to use cup. Attempts to feed himself with spoon.

3. *Food.*—Increase the variety of the foods given at 6 p. m. by adding creamed soup, spaghetti and so forth.

4. *Social Adjustment and Formation of Habits.*—Baby may indicate time for bowel movements. Discuss "no, no" stage in development; apply prohibition to one thing at a time.

5. *Physical State.*—Record anything noteworthy.

Explanatory Note Relative to Card 12.—Now that the child can get about readily, he wants to explore and to examine everything. Most things that he ought not to handle should be put out of his reach. Then he can be taught one thing at a time that he must leave alone, the most important things being dealt with first, such as the stove and the electric

light plugs. If he has his own corner and his own things to handle, often these can be substituted for those he cannot have. If "no, no" is said too frequently, it soon ceases to have any meaning.

LIMITATIONS AND USES OF THE CARDS

The cards are used, not to define rigid technics or instructions but to indicate approximately what can be expected of the average child at each period. Recognition of the wide variabilities of normal growth is necessary in their use. However, they do form a framework on which can be draped a child's emerging abilities, neuromuscular, social and personal.

MULTIPLE SCLEROSIS IN CHILDHOOD

H. R. CARTER, M.D.
Instructor in Neurology, University of Colorado School of Medicine
DENVER

MOST acute and chronic diseases found in young adults, such as tuberculosis, pneumonia, the allergies, undulant fever, meningoencephalitis, diabetes, nephritis, leukemia and Hodgkin's disease, are also found in common proportions in children. With the exception of syphilis of the central nervous system, multiple sclerosis is the most common primary disease of the central nervous system. Multiple sclerosis is a disease that is found in its classic forms, very commonly in young adults, just beyond the threshold of adolescence, yet it is rather uncommon in children. Many writers, Nobel,[1] Schupfer,[2] Bergen,[3] Wechsler,[4] Wilson,[5] Adie,[6] von Hoesslin[7] and Ford,[8] have spoken of its occurrence. The literature reveals a large number of cases, many of which are inadequately described. Only a few were followed for a period of years.

A number of statistical studies have appeared since the turn of the century. Wechsler's[4] study in 1922 summarized these past studies.

From the Division of Child Neurology, The Neurological Institute of New York and The Children's Hospital, Denver.

1. Nobel, E.: Ein Fall von multipler Sklerose bei einem 6 Jahre alten Kinde, Wien. med. Wchnschr. **62**:3231, 1912.

2. Schupfer, F.: Ueber die infantile Herdsklerose mit Betrachtungen über sekundäre Degenerationen bei disseminierter Sklerose, Monatschr. f. Psychiat. u. Neurol. **12**:60-70 and 89-120, 1902.

3. Bergen, A.: Eine Statistik über 206 Fälle von multipler Sklerose, Jahrb. f. Psychiat. u. Neurol. **25**:168-188, 1905.

4. Wechsler, I. S.: Statistics of Multiple Sclerosis, Arch. Neurol. & Psychiat. **8**:59-75 (July) 1922.

5. (*a*) Wilson, S. A. K.: Neurology, edited by A. N. Bruce, Baltimore, William Wood & Company, 1940, vol. 1, pp. 148-178. (*b*) Wilson, S. A. K., and Cottrell, S. S.: The Affective Symptomatology of Disseminated Sclerosis: A Study of One Hundred Cases, J. Neurol. & Psychopath. **7**:1-30 (July) 1926.

6. Adie, P. I.: Observations on Aetiology and Symptomatology of Disseminated Sclerosis, Brit. M. J. **2**:997-1000 (Dec. 3) 1932.

7. von Hoesslin, R.: Ueber multiple Sklerose: Exogene Aetiologie, Pathogenese und Verlauf, Munich, J. K. Lehmanns, Verlag, 1934.

8. Ford, F. R.: Diseases of the Nervous System in Infancy, Childhood and Adolescence, ed. 2, Springfield, Ill., Charles C Thomas, Publisher, 1945, pp. 324-329.

A review of this literature bears out his statements that the vast majority of cases are unproved, but why such erroneous cases were used by careful observers is difficult to understand. Wechsler reported 2 definite cases of multiple sclerosis which few would question, and stated: "Its existence, therefore, in children, while rare and in need of scrutiny, cannot be doubted."

With this in mind, a survey was made of some 711 cases diagnosed as multiple sclerosis at the Neurological Institute. This covered a period of eleven years, from 1932 to 1942 inclusive. Among the patients there were 2 girls aged 12 years at the onset of the disease (before the beginning of menstruation), 2 girls aged 13 (with one year of menstruation) and 1 boy aged 10 at the onset. Between puberty and 16 years of age there were ages at onset of 14 and 15, in 2 boys and 2 girls. Between the estimated ages at onset of 16 and 17, there was a sharp increase of cases, some 64 in number. The girls outnumbered the boys. The reason for this sharp increase is difficult to explain. This may have been noted by other workers, as von Hoesslin,[7] but most tables of age incidence (Marburg [9]) are given in decades, as in the table included.

Incidence of Multiple Sclerosis in Children

Source	Total Cases	Age Group	Number of Cases	Per Cent of Total
Neurological Institute	711	10 to 17	73	10.2
Wechsler [4]	194	1 to 20	12	6.2
Marburg [9]	131	1 to 20	25	19.8
Wilson [5b]	100	11 to 19*	14	14.0
Adie [6]	107	11 to 20*	11	10.3
von Hoesslin [7]	516	11 to 15†	21	4.0

* None reported under 10 years of age.
† After 16 years a great increase in frequency.

These studies show that in the greatest number of cases the patients have an age at onset of 21 to 30, with the 31 to 40 decade coming next.

Wilson [5a] reported seeing the condition in 2 girls each of whom was 9 years old at the onset of the disease. But these instances were not included in his study of 100 cases cited in the table.

SYMPTOMATOLOGY

In adults, multiple sclerosis presents a fairly clearcut picture of chronic multiple lesions of the central nervous system following a course of remissions and exacerbations. A wider scope of variations is revealed in the clinical symptoms of multiple sclerosis in children.

9. Marburg, O.: Multiple Sklerose (Encephalomyelitis periaxialis scleroticans disseminata), in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 13, pp. 551-553.

In the diagnosis of multiple sclerosis in the latter I believe that the same clinical diagnostic limitations should be used as for adults; namely, it should be thought of as a disease that is essentially afebrile with a chronic remitting course in which the varied components of the central nervous system may exhibit symptoms of disrupted function.

The onset in children as in adults shows several variations. The more sudden and fulminating onset is illustrated in the first case, while the slow insidious onset is shown in the second and third cases. The fourth case exemplifies a third type of onset, with an associated retrobulbar neuritis, as mentioned by Ford [8] and Adie.[6]

As the disease develops along its course, there may be noted rapidly developing central scotomas, exacerbation and remissions in visual acuity, transient diplopia, pallor of disks, spastic paraplegia, paraparesis, hemiplegia or monoplegia, loss of abdominal reflexes, ataxias, intention tremor, numbness, paresthesias, loss of vibration and position sense, alternations in speech, nystagmus, transient sphincteric disturbances, changes in emotional expressions, apathy, mild dementia and neurotic or hysterical manifestations. These symptoms are exhibited in the form of acute or subacute exacerbations. Remissions which follow are sometimes complete but usually partial.

REPORT OF CASES

CASE 1.—A white boy 10 years old at the time of onset had pain in the left leg and on the next day pain in the right leg with loss of control and some jerking movements of the legs and urinary frequency. He was admitted to Mount Sinai Hospital on May 10, 1942 with the following manifestations: a temperature of 101 F., tenderness over the spine from the sixth cervical to the seventh dorsal vertebra, stiffness in the gait, the abdominal reflexes absent except for slight response in the left upper quadrant, the deep reflexes decreased in the legs and a bilateral Babinski sign. There was a sensory level at the fifth and sixth dorsal vertebrae. Cerebrospinal fluid and roentgenologic examination revealed nothing abnormal. After a few weeks he made a recovery and played normally.

He was readmitted to Mount Sinai Hospital on June, 1943 with a history of numbness and weakness of the right upper extremity of some three weeks' duration. Examination showed weakness of the lower right side of the face, deviation of the tongue to the right and mild weakness of the right arm with pseudoathetosis. The right biceps reflex was slightly less active than the left. A questionable Chaddock sign was present on the right. Definite astereognosis with loss of vibration and position sense was noted in the right upper extremity.

On his admission to the Neurological Institute, Aug. 14, 1943, the patient showed a left hemiplegic gait, an extremely weak right arm, reflexes more active on the right than on the left, abdominal reflexes absent except for a slight response in the left upper quadrant and a bilateral Babinski sign. There was temporal pallor of the right optic disk. Ataxia of right arm, an increased tone in both lower extremities, a bilateral foot drop and a positive Romberg sign were present. When the right arm was held extended, there was a tendency for it to drift downward. The boy was able to perform poorly the right finger to nose test but was unable to put the left finger to the nose. Bilateral patellar and ankle

clonus, cremasteric and abdominal reflexes were absent except for a sluggish response in the right upper quadrant of the abdomen. Chaddock, Oppenheim and Gordon signs were present on the right side and there were bilateral Babinski signs. Vibration sense was absent bilaterally to the level of the internal malleolus. There were bilateral astereognosis and a scattered impairment of tactile and pain sensations. Slight pallor of the left optic disk and bilateral temporal scotomas were noted. The cerebrospinal fluid contained 14 cells per cubic millimeter, and the total protein content was 76 mg. per hundred cubic centimeters. On his discharge, August 26, the patient's left hand and arm were practically normal, with only pyramidal signs in the left foot.

One month later he had difficulty on micturition of one week's duration. During the winter, mild fluctuating pyramidal signs in the right and left legs were present, which were greater on the right, with some ataxia. He returned to school and engaged in all its activities.

In April 1944, he had an episode of some ten days' duration during which he was unable to grip well with his right hand. In July 1944 neurologic examination by Dr. Veronica O'Brien yielded results entirely within normal limits. One month later, numbness developed in the patient's left leg and foot, which disappeared after a week's duration. He then continued well until July 1945, when he started to drag his right foot. On August 2, Dr. O'Brien's examination showed a hemiparesis of the right side, weakness of the right central facial muscles and an increase of all tendon reflexes on the right side, with a stiffness of gait. Four days later there was a complete clearing of signs in the right arm and face. Pyramidal signs and ataxia in the right leg were more pronounced, and the tendon reflexes of the left lower extremity were exaggerated.

CASE 2.—A white girl was well until 9 years of age, when she had a sudden onset of aphasia following an attack of unconsciousness. Three days later her mother noticed that she dragged the right foot and was unable to use the right arm. After a few weeks this condition cleared to normal. At 14 years of age, she fell and lost consciousness for a few minutes. After this she had difficulty in the use of the left side of her body. This weakness of the left side continued with periods of remission and exacerbation. Later a tremor developed in the left arm, and the patient showed considerable emotional instability. A few months later she started to have difficulty with speech. This improved up to the time of the first admission to the Children's Hospital, Jan. 11, 1941. Here she was seen in neurosurgical consultation by Dr. C. G. Freed, whose examination revealed a 15 year old white girl somewhat below par mentally and frequently facetious. Convergent strabismus, nystagmus and some weakness of the left arm and leg were present. The left foot dragged, with adduction and internal rotation. The left thigh was slightly atrophied, and the left forearm was carried in a spastic position. A Babinski sign on the left and a tremor of the left hand were observed. There was a decreased cutaneous temperature of the left leg, with subcyanotic mottling. The tongue deviated to the left; the pharyngeal reflex was absent; the left triceps was hyperactive and the patellar and ankle tendon reflexes were hyperactive bilaterally. The blood and the cerebrospinal fluid were normal, and Mantoux tests elicited negative reactions.

During the next eight months, the patient continued to have progressive difficulty with her speech and gait, accompanied with some spasticity of the left side and emotional instability. In September 1941 she was unable to walk but showed improvement in speech and less spasticity in the left arm.

CASE 3.—A white girl, 12 years of age, had gradual development of weakness of the right side of the body and numbness in the right upper extremity. The next

day after feeling this numbness she found that she was unable to raise the right arm to shoulder level; her voice became thick and could be understood only with difficulty, and the right side of her mouth was weak, with the tongue pointed to the right. Six days later the right leg became weak and stiff. This paralysis varied from day to day. Her speech improved after two weeks.

On admission to the Neurological Institute, Feb. 3, 1932, the patient was observed to move the right leg stiffly and to drag the left foot. In her gait she deviated to the right and was unsteady, walking on her toes. Her right leg was spastic and of reduced strength. There was no gripping strength in the right hand, and there was slight asynergia of that hand. Her voice was thick and husky. The deep reflexes of the right upper extremity were more active than those of the left. Transient bilateral clonus of patellar and ankle reflexes, and crossed and uncrossed tibio-adductor reflexes were all active. Abdominal reflexes were absent on the right side, while those of the left lower portion of the abdomen were active, and those of the upper portion sluggish. Hoffmann, Babinski, and Chaddock signs were revealed on the right side. Examination of the eyes showed convergent strabismus. There was weakness of the muscles of the right central portion of the face and a lack of emotional expression on the right side. Movements of the uvula and the palate were weak on the right. The tongue deviated to the right, with some atrophy. After two months the right leg and the speech improved, and the child walked and talked without difficulty. Her right arm increased in strength, but the intention tremor of the right arm continued.

On her second admission to the Neurological Institute Nov. 1, 1932 examination showed hemiplegia on the left side, weakness in the left hand and dragging of the left leg. There was no grip strength in the left hand; the right grip was good. Intention tremor and athetoid movements of the right arm were observed. Her gait showed a bearing to the left. The right finger to nose test revealed an ataxic condition. It was impossible to test the left arm because of its extreme weakness. Patellar and hamstring reflexes were hyperactive. Abdominal reflexes were absent bilaterally, while Babinski and Oppenheim reflexes were elicited on both sides. Nystagmoid movements horizontally, residual weakness of the right central portion of the face and atrophy of the right side of the tongue were noted. By March 1, 1933, there was less tremor present, and the patient returned to school.

CASE 4.—A white girl 13 years of age, a patient of Dr. Irving Sands, gave a history of gradual weakness of the legs and of awkwardness, present since December 1941. In January 1942 she noticed decreased sensation in the right leg, followed by paralysis of the left leg and weakness of the left arm. Vision in the right eye was decreased to the extent that she was unable to read. There was an increasing weakness in the left hand. Other symptoms were urinary retention and incontinence.

Examination made on her first admission to the Neurological Institute, Feb. 16, 1942, revealed apathy, bilateral tremors of the hands and ataxia of the left upper extremity. The left hand exhibited mild contractures; the left leg, severe spasticity, and the right leg, moderate spasticity. All muscles of left upper and lower extremities were weak. The right finger to nose test showed some asynergia; the left, pronounced asynergia, which increased with closure of the eyes. Decided impairment was revealed in successive movements of the left upper extremity. The patient was poor at buttoning her clothes and was unable to repeat more than short sentences. Tests showed slight increase of the deep reflexes of the left pectoral, biceps, radial, triceps and wrist tendons. Sustained left patellar clonus and transient right patellar clonus was induced. The left ankle reflex was more

hyperactive than the right. All abdominal reflexes were absent. The Hoffmann sign was positive bilaterally and Babinski and Chaddock signs were elicited on the left side. Some decrease of response to tactile stimuli over the left hand was noted. There was a bilateral decrease of pain perception below the level of the sixth dorsal vertebra and also over the left arm and forearm. A bilateral absence of vibratory sense in the legs to the level of the fourth lumbar vertebra and a decreased vibratory sensibility in the hands and forearms were revealed and some decrease in the position sense for the distal parts of the extremities. Determination of the visual fields showed the child to have bilateral tubular vision. Pallor of optic disks was observed to be greater on the left than on the right side. Right internal strabismus, loss of convergence, inability to maintain a lateral gaze, nystagmoid jerking on a left lateral gaze and a decrease of the left corneal reflex were other optical conditions noted. The temperature varied between 99 and 100 F. and the pulse rate between 80 and 100. The reaction to the Kline test was negative. The cerebrospinal fluid contained 23 cells per cubic millimeter, all lymphocytes, and the protein content was 90 mg. per hundred cubic centimeters. An electroencephalogram, read by Dr. Paul Hoefer, showed an abnormal pattern, with focal signs in the motor and sensory areas.

Progressive increase of the sensory level to the fifth cervical vertebra by Feb. 21, 1942 was observed. The child was put into a respirator because of pain and weakness of the intercostal muscles. She was mentally sluggish, and her face was expressionless; her tongue deviated to the left. By March 31, 1942, the patient's electroencephalogram had assumed a more normal pattern. She was able to take a few steps. On April 17, spontaneous jerking of both arms, greater in the right than in the left, developed. Vision suddenly blurred, and she was just able to perceive light. Her pulse rate was 150, with a moderate rise in temperature and a sensation of chilliness. There was increased weakness in the left lower extremity and in the grip of the left hand. The finger to nose test showed greater incoordination on the left than on the right side. The left heel to knee test revealed definite incoordination. The deep reflexes were hyperactive, with a greater increase on the left side than on the right and bilateral ankle clonus was elicited, the response of the left side being greater than that of the right. Plantar reflexes were absent bilaterally. On examination of the eyes poor convergence, nystagmoid jerking on lateral gaze and temporal pallor of the right optic disk were observed. Three weeks later the patient showed definite improvement, with normal visual fields except for a slight increase in the blindspots. This progress continued, and she was able to walk unassisted. There was some weakness in left upper extremity, with the right hand showing clumsiness, and the child was unable to recognize objects.

The patient was admitted to the Neurological Institute for a second time, June 10, 1942. An examination showed the presence of ataxia of the left arm and foot, a pronounced Romberg sign, inability to write and a slight increase of the deep reflexes bilaterally; abdominal reflexes were absent. Vibratory sense was absent from the xiphoid process down. Scotomas of the right eye and bitemporal pallor of the optic disks were observed. An intermittent course necessitated a third and fourth admission to the Neurological Institute. When the patient was admitted for the fifth time, Oct. 22, 1944, the right hand was numb and weak and there was inability to control its movements. The patient felt a heavy sensation in the left leg and dragged it. She revealed emotional instability and had laughing and crying spells. There was a loss of automatic movement in the right upper extremity. The Romberg sign was present in a slight degree. Tests of the finger reflexes

showed a greater increase in those of the right fingers than in those of the left. The patellar reflexes were of a pendular type, the left being extremely hyperactive. Abdominal reflexes were absent bilaterally. Babinski and Chaddock signs were elicited on the left side. The left eye had three scotomas in the 12 o'clock area, and there was bitemporal pallor of the optic disks. A slight flattening of the left side of the face was noted. After three weeks the gait and the movements of the right arm became normal.

DIFFERENTIAL DIAGNOSES

In other cases after the onset the disease may develop acutely within a few days, with mild fever, stiffness of the neck, convulsions, mental confusion or partial coma, disseminated focal signs and an increased cell count and protein content of the cerebrospinal fluid. In these cases the condition resembles acute disseminated encephalomyelitis and cannot be differentiated from it early in the disease. Recovery from this acute phase is followed by future exacerbations and remissions. This seems to remove it from the acute disseminated encephalomyelitis which follows vaccination against variola, measles, mumps, scarlet fever and other infectious diseases. The latter conditions fail to have a subsequent course of exacerbations and remissions. The symptoms of acute disseminated encephalomyelitis are often initiated by an influenza-like illness, fever, headache, muscle pains in the back and vomiting. These manifestations are followed within a few days by restlessness, irritability, drowsiness, stupor, diplopia, paralysis or paresis and retention of urine. These symptoms are of short duration. This relationship of multiple sclerosis and acute disseminated encephalomyelitis is described by the French writers, Rimbaud, Riser and Geraud,[10] van Bogaert [11] and others. The clinical observations in their cases follow those of acute disseminated encephalomyelitis seen in children, the majority of patients having a moderate febrile onset, with convulsions. Cerebellar symptoms are frequent, with the persistence of the abdominal reflexes and the lack of remissions and exacerbations observed in multiple sclerosis. Wilson [5a] expressed the belief that enduring sequelae were rare when the condition took place in childhood. In the differential diagnosis, he further stated that the pain, fever and meningeal reaction do not on the whole find a counterpart in acute examples of polysclerotic disease but that they are at times minimal and belong in reality to polysclerotic disease. The histopathologic

10. Rimbaud, L.; Riser, and Geraud: De la sclérose en plaques chez l'enfant, Rev.neurol. **69**:477-481 (May) 1938.

11. van Bogaert, L.: Contribution à l'étude de la sclérose en plaque chez l'enfant, Ann. de méd. **31**:59-79 (Jan.) 1932.

picture alone is not sufficient, as it is produced by a number of different etiologic factors. The diagnosis can be made only by postmortem examination and by virus and animal transmission studies.[12]

Another type of onset in children is that with a retrobulbar neuritis of one or both eyes, in which there are rapidly developing central scotomas and a loss of visual acuity. A short or long period of remission is followed by an exacerbation of spastic paraplegia and ataxia. This condition is closely related to neuromyelitis optica, occurring often in childhood and adolescence, in which there are atrophy of the optic nerve and lesions of a transverse myelitis. Ferraro[13] stated that acute multiple sclerosis, acute disseminated encephalomyelitis, and neuromyelitis optica cannot be differentiated histopathologically. This observation is equally true for the clinical experience of most neurologists.

Leber's optic atrophy has strong hereditary trends, onset at puberty (mostly in boys) and predominance of ocular symptoms.

Hall and Mackay[14] emphasized the difficulty of the differential diagnosis of multiple sclerosis in cases of familial cerebellar ataxias, Marie's cerebellar ataxia and Friedreich's ataxia. In the familial cerebellar ataxia, olivopontocerebellar atrophy is in evidence, the onset is in early life, there is an absence of cord involvement, deep and superficial reflexes are present, and there is no Babinski sign or impairment of the vibratory sense. The principal observations are advance nystagmus, scanning speech and intention tremor. Marie's cerebellar ataxia shows greater variation, a slow spread of walking and standing difficulties, with nystagmus, atrophies and ophthalmoplegias of varying degrees. The Babinski sign may occur with increased deep reflexes. Friedreich's ataxia has an onset in early age, with difficulty in walking, pes cavis, scoliosis, slurred explosive speech, action tremors, nystagmus and muscular weakness. Deep reflexes may or may not be present. Control of the bladder and bowels may be impaired. Finally, there is an unremitting course downward.

Tumors of the brain stem will often produce confusing and scattered effects suggestive of multiple sclerosis. A number of patients whose disease was diagnosed initially as multiple sclerosis were found on subsequent examinations to have such tumors.

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12. Grinker, R., and Levy, N. A.: Neurology, ed. 3, Springfield, Ill., Charles C Thomas, Publisher, 1943, chap. 15, pp. 811-823.

13. Ferraro, A.: Primary Demyelinating Processes of the Central Nervous System: An Attempt at Unification and Classification, Arch. Neurol. & Psychiat. **37**:1100-1160 (May) 1937.

14. Hall, G. W., and Mackay, R. P.: Forms of Familial Ataxia Resembling Multiple Sclerosis: A Clinical Study, Arch. Neurol. & Psychiat. **37**:19-32 (Jan.) 1937.

TREATMENT

An attitude of rejection in therapy is easy to develop in regard to multiple sclerosis both in adults and in children. The rapidly changing neurologic picture in case 1 well illustrates how difficult any evaluation of therapy might be. In cases 2 and 3 the patients were given supportive physical therapy and arsenicals. In case 4 the patient was given dicoumarin (3,3'-methylene-bis-[4-hydroxycoumarin]) during the acute phase of the illness, with an average undiluted prothrombin time of twenty-six seconds. This level was more or less maintained during the three year period during which she was continuously given this drug. During this period the patient had some four exacerbations and remissions, necessitating readmissions to the hospital. Some of the more recent therapeutic attempts have included the use of hemolytic serum,[15] lecithin,[16] thiamine intrathecally and pyridoxine,[17] trypan red, intravenously,[18] dicoumarin,[19] histamine intravenously[20] and change of habitation[21] to a climate warm the year around. The use of fever therapy is still a subject for argument. Some patients have shown a striking and rather long remission following its use, while other patients were made worse. The approach to effective therapy will be by a greater knowledge of the biochemical and immunologic factors. This progress is slow and must await research in the fields of the basic medical sciences. So pressure comes from clinical necessity to try empirically certain methods of treatment which may have a plausible approach, in the hope that some effective therapy may be developed and the problem be worked backward. Such a course is frequently

15. Beck, P., and Martini, P.: Nachprüfung der Behandlung der multiplen Sklerose mit hämolytischem Serum (Laignel-Lavastine und Koressios), Nervenarzt **13**:103-117 (March) 1940.

16. Minea, I.: Ueber die Lezithinbehandlung der multiplen Sklerose, München. med. Wchnschr. **86**:1038-1040 (July 7) 1939.

17. Stone, S.: Subarachnoid Administration of Pyridoxine Hydrochloride in Diseases of the Nervous System, J. Nerv. & Ment. Dis. **100**:185-190 (Aug.) 1944.

18. Aird, R. B., and Strait, L.: Protective Barriers of the Central Nervous System: An Experimental Study with Trypan Red, Arch. Neurol. & Psychiat. **51**:54-66 (Jan.) 1944. Aird, R. B.: Personal communication to the author, Sept. 3, 1945.

19. Reese, H. H.: Multiple Sclerosis and Dicumarol Therapy, Tr. Am. Neurol. A. **70**:78-84, 1944.

20. Horton, B. T.; Wagener, H. P.; Aita, J. A., and Woltman, H. W.: Treatment of Multiple Sclerosis by the Intravenous Administration of Histamine, J. A. M. A. **124**:800-801 (March 18) 1944.

21. Freeman, W.: Frontiers of Multiple Sclerosis: Treatment of Multiple Sclerosis, with Special Reference to Fever Therapy, Prolonged Rest and Climatotherapy, M. Ann. District of Columbia **13**:58-67 (Feb.) 1944.

followed in clinical medicine. Despite all such attempts, therapy seems no more effective than that at the turn of the century. In the meantime, the clinician must continue to depend on empiric medical treatment of this disease while waiting for research in the fields of biochemistry and immunology.

COMMENT

Ferraro [22] has correlated the histopathologic changes found in multiple sclerosis and other demyelinating diseases with those found in acute, subacute and chronic allergic reactions. The same histopathologic conditions were caused by factors completely unrelated to the demyelinating diseases. Even though the etiologic factors are diverse, the common tissue hypersensitivity mechanism seems to be in evidence. This tissue hypersensitivity is possibly an immunity reaction to the etiologic factor, a reaction which for the most part is maintained at a high level of resistance, as noted in periods of remission, and is temporarily lowered in periods of exacerbations. The rarity of multiple sclerosis in children leads one to surmise that the immunity to it is probably held high in infancy and childhood, except when these etiologic factors overwhelm the organism, as seen in the diffuse sclerosis of early childhood. On the threshold of maturity, there may be a temporary instability of the immunity factors. What changes take place in the gamma globulin fractions following puberty cannot at this time be even postulated. There may remain a focus of an antigenic character which becomes active and inactive according to fluctuation in immunity and in hypersensitivity of the nervous tissue. The spread from this antigenic focus to the hypersensitive nervous tissue may be hematogenous. Myelin tissue of the nervous system is mostly affected, damage to the axon being less in evidence. When the myelin anabolism and catabolism are no longer in a reversible phase, then progressive gliosis ensues. Acute demyelinating lesions will develop in the same tissue in which chronic sclerotic plaques have been in existence for some time. That these concepts explain the remissions and exacerbations is not proved. It is thought that in the repeated hemiplegic picture (case 1) the myelin tissue must be capable of reversible phases of anabolism and catabolism.

Whether the initial etiologic factor is a virus-like antigenic focus with resultant changes in the globulin immunity mechanism resulting in demyelination is uncertain. And whether there is an enzyme having a myelinolytic action, which, possibly elaborated by the liver metabolism, passes the blood-brain barrier to act in a diffuse or disseminated manner

22. Ferraro, A.: Pathology of Demyelinating Diseases as an Allergic Reaction of the Brain, Arch. Neurol. & Psychiat. **52**:443-483 (Dec.) 1944.

on nervous tissue, is equally uncertain. Brickner [23] made studies in search for an abnormal lipase in the blood of patients with multiple sclerosis, his supposition being supported by his observation that oxalation of lecithinated serum and plasma of patients with multiple sclerosis inhibits acid production much more than is normal. Weil and Heilbrunn [24] reported the demonstration of a myelolytic substance in the urine of patients with multiple sclerosis after observing that both their urine and a solution of sodium taurocholate acted on the myelin sheaths of the spinal cords of rats causing an appearance of granulation but that the urine of normal controls and that of patients with central nervous system syphilis, subacute combined degeneration and other degenerations of the brain and spinal cord failed to break up the sheaths in a similar manner, showing the absence of the myelolytic substance. Besides multiple sclerosis, postencephalitis and certain hepatic diseases caused the same intensive myelin destruction. Richards and Wolff [25] could find no method to measure the hydrolysis of such a complicated substance as myelin. Their findings were to the effect that serum, cerebrospinal fluid and plasma from patients with multiple sclerosis yielded no different results than fluids from the normal controls in the action on the spinal cords of white rats and rabbits. The use of trypan red, a supravital diazo dye, in altering the permeability of the blood-brain barrier, so that toxic and degenerative factors pass through less readily, has been suggested by Aird and Strait.[18]

In children it is difficult to find the factors common to both sexes that give them a high resistance to multiple sclerosis, a resistance which may be lowered within a short few years following puberty. Why this occurs is unknown, and when this question can be answered possibly the key to the demyelinating diseases may be near at hand. That the mechanism causing multiple sclerosis is on the basis of allergic tissue hypersensitivity is difficult to understand in the light of the present knowledge of allergies, which are found in such abundance from infancy to adulthood. The short and transitory remissions and exacerbations of the symptoms can be readily thought of as being due to transitory allergic tissue edema which interrupts function of the central nervous system temporarily, and that permanent loss of function comes only after prolonged or repeated episodes of tissue hypersensitivity.[26]

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23. Brickner, R.: Studies of Pathogenesis of Multiple Sclerosis: Evidence of the Presence of an Abnormal Lipase in the Blood in Multiple Sclerosis, Bull. Neurol. Inst. New York **1**:105-135 (Jan.) 1931.

24. Weil, A., and Heilbrunn, G.: Demonstration of Myelolytic Substances in Disseminated Sclerosis, Proc. Soc. Exper. Biol. & Med. **48**:233-237 (Oct.) 1941.

25. Richards, C. H., and Wolff, H. G.: Studies in Multiple Sclerosis: Serum Enzymes, Arch. Neurol. & Psychiat. **43**:59-69 (Jan.) 1940.

26. Kennedy, F.: Allergy and Its Effect on the Central Nervous System, J. Nerv. & Ment. Dis. **88**:91-105 (July) 1938.

SUMMARY

A recent statistical study of the incidence of multiple sclerosis in childhood has been made, showing its relative infrequence. Four illustrative cases showing the variations in onset and symptomatology are described. The differential diagnosis, with particular mention of childhood diseases of the nervous system is presented. Therapy for children with multiple sclerosis is at the same impasse as that for adults. Certain concepts as to pathogenesis have been postulated.

Dr. Louis Casamajor, Chief of the Division of Child Neurology, The Neurological Institute, New York, aided in this study and Dr. Irving Sands and Dr. Veronica O'Brien gave me an opportunity to study their patients.

550 Metropolitan Building.

CONGENITAL BILIARY CIRRHOSIS

IRVIN DUNSKY, M.D.
CINCINNATI

IN DISTURBANCES of carbohydrate metabolism it is not uncommon for hyperglycemia and glycosuria to be interpreted as diabetes mellitus, while the extrapancreatic melliturias are overlooked. It seems worth while, therefore, to report an instance of hepatic diabetes occurring prominently in association with a rare case of biliary cirrhosis. The unusual features of hepatic diabetes associated with congenital biliary cirrhosis without obstruction of the extrahepatic bile ducts and with concomitant renal involvement, are, accordingly, considered. The value of the intravenous dextrose tolerance test [1] in differentiating between pancreatic and hepatic diabetes is also discussed.

REPORT OF A CASE

The patient, R. K., admitted to The Children's Memorial Hospital at 2 months of age, was the product of a full term, normal pregnancy. His birth weight was 6 pounds 13 ounces (3,090 Gm.). He was breast fed for only the first four days of life, at which time jaundice became apparent. A week later jaundice still persisted, although the color was somewhat lighter. The infant was extremely irritable and took an evaporated milk formula poorly. Urinalysis, done at that time and repeated frequently thereafter, revealed a constant albuminuria, hyaline and granular casts and a 3 plus reaction for sugar. At 6 weeks of age, enlargement of the abdomen was first noted, together with a persisting jaundice. Abdominal enlargement was progressive and prompted hospitalization.

The parents and one sibling were living and well. There was no history of maternal dietary deficiencies during the course of the pregnancy. There was no history of syphilis, tuberculosis, malaria or alcoholism and no known exposure to or ingestion of toxic substances. Pregnancy had been normal and uneventful, and no blood transfusions had been given to the mother. The family history, however, was interesting. The mother's mother and grandmother had both been diabetic. Of the mother's four siblings, one died of diabetes, a second "from jaundice at 1 week of age," while a third, aged 29 and living, suffered from "gallbladder trouble." The mother herself had one episode of jaundice (catarrhal?) at the age of 7 but, except for a repaired congenital cleft palate, had otherwise always been well. The father's mother had diabetes, but there was nothing suggestive of a hepatic disorder in his family.

Physical examination at the time of admission revealed an irritable, malnourished white boy weighing 7 pounds 8 ounces (3,402 Gm.). The skin and scleras were mildly icteric, and several petechial hemorrhages were noted in the

From The Children's Memorial Hospital, Chicago.

1. Soskin, S.: Role of the Endocrines in the Regulation of Blood Sugar, J. Clin. Endocrinol. 4:75, 1944.

right axilla. Telangiectasia and dilated superficial veins were noted in the skin of a moderately distended abdomen. Spider angiomas and a caput medusa were not seen. There was no palmar erythema, clubbing of the fingers or curving of the finger nails. The chest was clear. An apical murmur, interpreted as functional, was heard. A firm mass was felt extending 1½ fingerbreadths below the right costal margin, but the spleen was not palpable. A fluid wave was present. An abdominal paracentesis was done, and 300 cc. of a thin, clear, light yellow fluid with a specific gravity of 1.010 was obtained.

Laboratory Observations.—The blood count was normal. Reactions to Wassermann, Kahn, and Eagle tests on the patient and his mother were negative. A Mantoux test (1 : 1,000) elicited a negative reaction. On repeated analyses, the urine contained albumin (2 plus), sugar qualitative, 3 plus; quantitative, 1.5 mg. per hundred cubic centimeters, and numerous granular and hyaline casts. Acetone, bile and urobilin were not found in the urine. A fermentation test of the reducing substance in the urine showed its presence in a 0.1 per cent concentration after eighteen hours. The patient and the mother were both Rh positive.

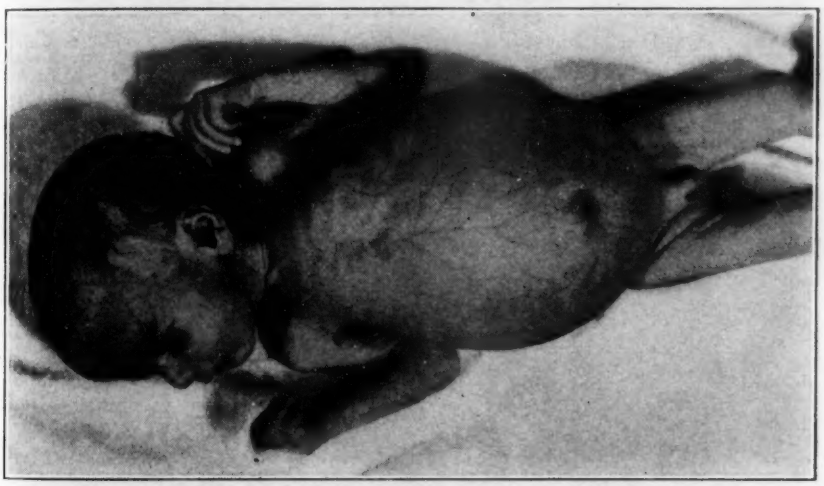

Fig. 1.—The patient, R. K., at 2 months of age.

Other observations were as follows: The icterus index was 25; the cephalin flocculation reaction, 4 plus; cholesterol level, 148 mg. per hundred cubic centimeters (cholesterol esters, 21 per cent of the total cholesterol); total protein level, 3.8 Gm. (albumin 2.4 Gm., globulin 1.4 Gm. and albumin-globulin ratio 1.7); prothrombin time, 85 per cent of normal; fibrinogen, 0.4 Gm. per hundred cubic centimeters; calcium, 9.9 mg., and bleeding and clotting times, normal. A six hour fasting blood sugar level was 238 mg. per hundred cubic centimeters. Soskin's (six hour), intravenous dextrose tolerance test [1] revealed the following capillary blood sugar levels after fasting, and in one-half hour, one and two hours respectively: 205, 235, 207 and 177 mg. per hundred cubic centimeters. An epinephrine tolerance test,[2] using ½ minim (0.03 cc.) of a 1 : 1,000 solution of epinephrine per kilogram, revealed the following blood sugar values after fasting (six hours), and in one-half

2. Holt, L. E., Jr., and Bridge, E.: Hypoglycemia in Early Life, Nebraska M. J. **30**:45, 1945.

hour, one hour and one and one-half hours, respectively: 210, 188, 192 and 205 mg. per hundred cubic centimeters. The blood urea nitrogen on several determinations was 30 mg., the nonprotein nitrogen 60 mg. and the creatinine 1.5 mg. per hundred cubic centimeters. The culture of the ascitic fluid yielded no growth. The protein content of various samples of ascitic fluid obtained by weekly paracentesis ranged from 328 to 475 mg. per hundred cubic centimeters. Unchanged bilirubin (2 plus) was found in the stools.

Course.—The infant followed a progressively downhill course despite all measures. Omentopexy was considered but seemed inadvisable. The patient died three weeks after admission. The clinical impression was that of cirrhosis of the liver, hepatic diabetes and toxic nephrosis.

Autopsy.—The significant gross pathologic change was limited to the liver. All other organs and tissues, including the pancreas, the adrenal glands, the thyroid gland and the pituitary glands, were grossly normal.

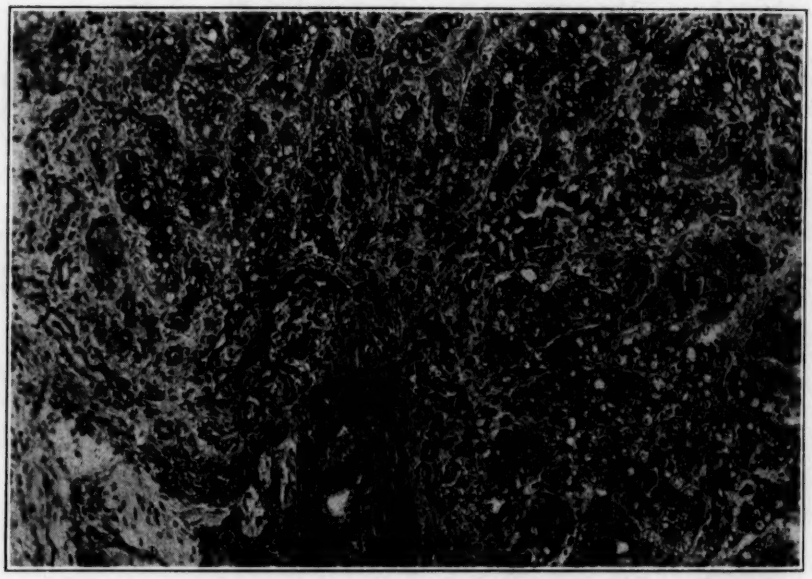

Fig. 2.—Liver (× 145). Stained with hematoxylin and eosin. Note (1) the diffuse increase of fibrous tissue which extends outward from the triads between and into the lobules; (2) the numerous small bile ducts in the bands of fibrous tissue; (3) the masses of inspissated bile in bile capillaries, and (4) the swelling and vacuolation (fat) of the hepatic cells.

The liver was bright green and firm to palpation and weighed 124 Gm., which was below the normal (140 Gm.) for the patient's age. The external surface was finely granular. The organ cut with increased resistance. The cut surface was a firm gray-green with indistinct lobular markings. The gallbladder was in its normal position and contained 2 cc. of a light yellow fluid. The cystic and hepatic ducts were carefully dissected and were found to be patent and normal.

Significant microscopic pathologic changes, limited to the liver and kidneys, were described by Dr. James P. Simonds as follows:

Examination of sections of the liver reveals a complete derangement and distortion of the histologic architecture. Intercommunicating bands of immature

connective tissue enclose irregular masses of hepatic cells. These cells are arranged more frequently in groups than in cords, and the central vein, if present, is usually at the margin of a mass of hepatic cells.

Within the bands of immature connective tissue are innumerable ductlike structures, which vary in appearance. Some are lined with tall columnar epithelium and have wide empty lumens. Less numerous are smaller ducts lined with cuboidal epithelium. The lumens of these smaller ducts are absent or small and are filled with a homogeneous mass of brown material. Innumerable inflammatory cells—monocytes, lymphocytes and polymorphonuclear leukocytes—are also present in the bands of the connective tissue. The bile ducts and the connective tissue make up almost half of the entire section.

The hepatic cells are much larger than normal, and their outlines are indistinct. In many places these cells are fused into a syncytium with the nuclei widely separated. Many of the large cells are vacuolated. Some of these vacuoles are

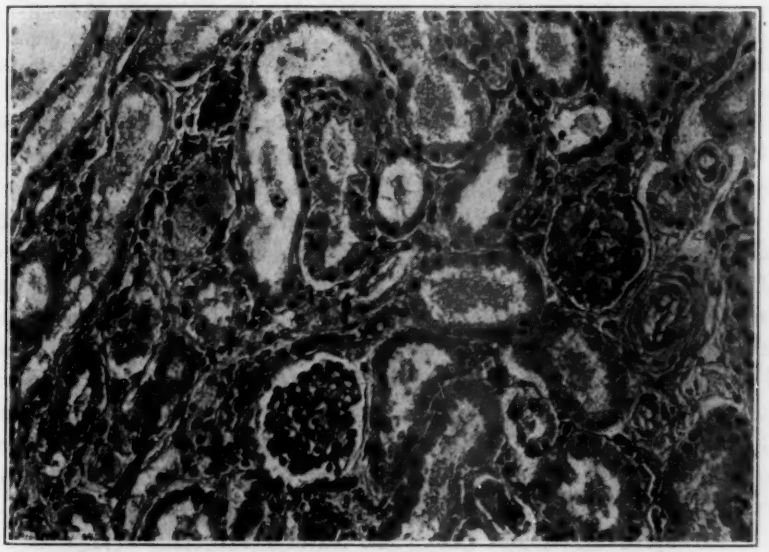

Fig. 3.—Kidney (× 145). Stained with hematoxylin and eosin. Note (1) the large size and increased cellularity of the glomeruli, which completely fill the capsular spaces; (2) the granular (precipitated albuminous) material in the dilated proximal convoluted tubules, and (3) the calcium deposit (a black pointed mass near the upper left margin).

clear; others contain a finely granular brown material. Search was made for masses of purple-staining hyaline material described by Mallory as characteristic of alcoholic cirrhosis, but none were found.

Examination of sections of the kidneys shows glomeruli that are large and completely fill their capsular space. Their large size is due to an increase in the endothelial cells lining the glomerular capillaries. Many of the glomeruli contain coagulated granular material in their capsular spaces. The proximal convoluted tubules are dilated and contain a granular material. The cells lining these tubules are flattened, larger than normal and often lacking in nuclei. Deeper in the cortex and in the outer zone of the medulla, many tubules are filled with a coagulated material resembling casts. In a considerable number of the tubules

there are numerous dark purple masses resembling calcium deposits. Large mononuclear cells and small giant cells lie against the surface of these masses. The walls of the smaller arteries are thicker than normal. The lesion is inflammatory and is both exudative and proliferative. The last-named characteristic is made evident by an abundance of young connective tissue, while the first-mentioned one is manifested by the great number of inflammatory cells lying in the new connective tissue.

The extensive destruction of hepatic tissue noted probably began in utero at a time when the bile ducts and hepatic cells still had strong regenerative power. This is shown by the great proliferation of bile ducts. The regeneration of hepatic tissue has not followed an orderly histologic pattern. Many of the regenerating cells have failed to divide completely, giving rise to the syncytial masses mentioned. Sections of the kidneys reveal (1) a glomerulonephritis, characterized by a proliferation of the endothelium lining the capillaries and by the presence of coagulated albuminous material in the capsular spaces, and (2) a pronounced parenchymatous degeneration and even necrosis of many of the cells lining the proximal convoluted tubules. The protein which has escaped through the glomeruli and the debris from disintegrated renal cells are the sources of the granular material found in the lumen of these tubules. Many of the casts in tubules formed from this material have become calcified, thus acting as foreign bodies.

The pathologic picture is that of a biliary cirrhosis, with associated renal involvement.

COMMENT

Cirrhosis of the liver in the young is a relatively rare, infrequently considered disease entity. Morse [3] estimated it to occur in 1 of every 20,000 children. Mallory [4] found 11 instances among 2,066 autopsies done on children in the first two decades of life, while Rolleston [5] found only 12 instances among some 16,000 autopsies done on patients under 21 years of age. Moon,[6] however, indicated that its occurrence was underestimated, citing 320 cases reported by Seitz [7] and adding some 90 cases collected from the literature. Notwithstanding the moot question of exact occurrence, cirrhosis in childen is well defined in its clinical manifestations and pathologic features, though the pathogenesis is often obscure. Various prominent features especially related to the disease in childhood have been emphasized by different observers.[8] The

3. Morse, J. L.: Cirrhosis of the Liver in Childhood, Boston M. & S. J. **147**:299, 1902.

4. Mallory, F. B.: Cirrhosis of the Liver, New England J. Med. **206**:1231, 1932.

5. Rolleston, H., in Christian, H. A.: Oxford Medicine, New York, Oxford University Press, 1936, vol. 3, p. 384.

6. Moon, V. H.: Atrophic Cirrhosis in Children, Am. J. Dis. Child. **46**:375 (Aug.) 1933.

7. Seitz, C., in von Pfaundler, M., and Schlossman, A.: Handbuch der Kinderheilkunde, Leipzig, F. C. W. Vogel, 1924, vol. 3, p. 359.

8. (*a*) Poynton, F. G., and Wyllie, W. G.: Hepatic Cirrhosis in Children, Arch. Dis. Childhood **1**:1, 1926. (*b*) Ladd, W. E., and Gross, R. E.: Abdominal Surgery in Childhood, Philadelphia, W. B. Saunders Company, 1941, p. 264.

familial incidence of cirrhosis in the young is often recorded. The occurrence of hepatic disorder in various relatives of the patient whose case is being reported has, accordingly, been noted. It is unlikely, however, that these represented examples of unrecognized familial hepatic cirrhosis, for Rössle [9] found instances of familial occurrence in successive generations distinctly exceptional. Hereditary familial cirrhosis manifests itself, by and large, in the same generation and among siblings. Juvenile cirrhosis, more common in boys than in girls, is more acute in its onset and shorter in its course than in adults. Moon [6] reported a case in which death occurred ten weeks after the onset of symptoms. Epistaxis is frequent and telangiectasis often present, though ascites and dilatation of the superficial vessels are often absent.[8a] There may be little or no digestive disturbance.[8b] Infantilism and bilateral osteoarthropathy are occasional special features in children.[10] Hemorrhage is common and often profuse. "About one third or more of them bleed with unusual ease, sometimes from known trauma and sometimes apparently spontaneously." "Unlike the patients with severe degrees of neonatal jaundice of haemolytic origin, who seldom, if ever, exhibit either itching of the skin or haemorrhage, the infants with jaundice due to congenital malformation of the bile ducts are prone to show one or both of these signs." [11] The spleen is involved in 90 per cent of the cases of juvenile cirrhosis,[6] and the liver is regularly enlarged, because of the acute course, death occurring before atrophy of the liver takes place.

Whereas 90 per cent of adults dying of cirrhosis exhibit the portal form,[6] cirrhosis of the liver in infants is more often biliary than portal in type. Acquired obstructive biliary cirrhosis, Hanot's hypertrophic cirrhosis (acquired, nonobstructive), infantile biliary cirrhosis of India and congenital biliary cirrhosis have been recognized [8d] as the four main types of biliary cirrhosis. Congenital biliary cirrhosis is usually associated with, but does not necessarily result from, obstruction of the extrahepatic bile ducts. Cases of congenital biliary cirrhosis without obstruction of the bile ducts have been described, and this condition is clinically indistinguishable from that in which there is ductal obliteration.

(*c*) Calvin, J. K., and Saffro, L. B.: Cirrhosis of the Liver in Children, Am. J. Dis. Child. **43**:914 (April) 1932. (*d*) Evans, P. R.: Biliary Cirrhosis, Arch. Dis. Childhood **14**:89, 1939. (*e*) Moon.[6]

9. Rössle, R.: Antzündungen der Leber, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1930, vol, 5, p. 274.

10. Levy, W.; Greenstein, N. M., and Leighton, B.: Portal Cirrhosis in Childhood, J. Pediat. **15**:91, 1939. Calvin and Saffro.[8c]

11. McIntosh, R.: Jaundice as a Pediatrician Sees It, Canad. M. A. J. **45**:488, 1941.

Poynton and Wyllie,[8a] citing 2 examples, mentioned a case without obliteration of the bile ducts, in which jaundice was first noted at the end of the patient's third week of life, and a second case, with ductal obliteration, in which jaundice was noted when the patient was 7 weeks old. Of the 22 cases of congenital biliary cirrhosis found in the post-mortem records of the Children's Hospital at Great Ormond Street,[8a] patent ducts were noted in 7. The apparent rarity of the condition was indicated in the American literature by Munns,[12] who reported a similar condition first manifested by jaundice in an infant 18 days of age. Munns was able to find only 2 closely similar cases.[13] Parsons and Hickmans [14] reported a case of atresia of the intrahepatic bile ducts, but without atresia of the extrahepatic bile ducts, in a girl in whom jaundice developed after three weeks of life and who died at 6 months of age. In the case now being reported, jaundice was first noted on the patient's fourth day of life. While it may well have been an expression of physiologic jaundice at that time, evidence of hepatic insufficiency was manifested by a disturbance in carbohydrate metabolism in the second week of life, with jaundice persisting until death. This case of congenital cirrhosis lends little support to the theory that biliary cirrhosis necessarily follows a developmental defect of the bile ducts, for it was Thomson's [15] view that a developmental aplasia of the bile ducts precedes hepatic cirrhosis in the young. Ylppö [16] pointed out, further, that the extrahepatic bile ducts patent in early fetal life lose their lumens by an epithelial concrescence, which is followed by a subsequent vacuolation. An arrest of development during the solid stage was suggested as the explanation for the congenital atresia. In view of the occurrence of biliary cirrhosis in the absence of ductal obliteration, one seems warranted in concluding that the underlying cause may be primarily within the liver itself. Rolleston and McNee,[17] accordingly, regarded the cirrhosis as primary, with fibrosis and obliteration of the larger ducts secondary to a descending cholangitis. Toxic substances are presumed to enter the fetal circulation, passing in part directly to the liver and in part directly to the general circulation via the ductus

12. Munns, G. F.: Chronic Interstitial Hepatitis, Am. J. Dis. Child. **31**:469 (April) 1926.

13. Dunn, C. H.: Cirrhosis of the Liver, Interstate M. J. **23**:14, 1916. Porter, L., in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1925, vol. 3, chap. 52, p. 656.

14. Parsons, L. G., and Hickmans, E. M.: Biliary Cirrhosis, Am. J. Dis. Child. **31**:459 (April) 1926.

15. Thomson, J.: On Congenital Obliteration of the Bile Ducts, Edinburgh M. J. **37**:523, 1892.

16. Cited by Ladd,[8b] p. 263.

17. Rolleston, H. D., and McNee, J. W.: Diseases of the Liver, Gallbladder and Bile Ducts, ed. 3, London, The Macmillan Company, 1929.

venosus, reaching the liver by the hepatic artery, which supplies the interlobular connective tissue and its contained structures. A mixed cirrhosis, as seen in this case, may thus obtain, which may progress and give rise to a descending obliterative cholangitis.

Mellituria, first noted in the second week of life and regarded as glycosuria, was an early manifestation in this case of a disturbed carbohydrate metabolism, subsequently shown to be due to hepatic dysfunction. Considering the fact that hepatic activity remains apparently normal as long as 15 per cent of the total hepatic tissue retains its anatomic integrity and that in the case of some functions even 5 per cent of undamaged tissue suffices,[18] one is forced to assume a process of relatively long duration and thus, of necessity, intrauterine. That the presence of a reducing substance in the urine, in general, must be considered critically becomes apparent when it is realized that 1 of every 6 persons with a mellituria who is suspected of having pancreatic diabetes is ultimately proved to have an extrapancreatic condition.[19] Glycosuria per se is not evidence of a hyperglycemia unless the renal threshold is known, and hyperglycemia per se is not evidence of pancreatic disease unless extrapancreatic mechanisms are considered. That the liver must be considered seriously hardly needs elaboration, for the liver is the central, homeostatic mechanism of the body for storage of glycogen and supply of glucose. Hepatic dysfunction was indicated in our case by clinical signs of jaundice, hepatomegaly, ascites and petechial hemorrhage. Laboratory observations, including an elevated icterus index, a 4 plus cephalin flocculation reaction, low level of cholesterol esters and hypoproteinemia with hypoalbuminemia, supported the clinical impression. It seemed reasonable to assume that the high blood sugar level and persistent mellituria were either the result of delayed assimilation of sugar by the liver or an expression of the impairment of the normal glycogenolytic inhibition of the liver to the stimulation of exogenous glucose.[1] The history of diabetes in so many members of the family, however, introduced a note of caution, for pancreatic diabetes was yet possible, particularly so in view of the high blood sugar level. Indeed, Joslin[19] has stated that a capillary blood sugar level above 200 mg. per hundred cubic centimeters justifies a diagnosis of pancreatic diabetes. Moreover, the simultaneous occurrence of hepatic and pancreatic cirrhosis has often been observed.[20]

18. Soffer, L. J.: Present Day Status of Liver Function Tests, Medicine **14**: 185, 1935.

19. Joslin, E. P.: Treatment of Diabetes Mellitus, ed. 6, Philadelphia, Lea & Febiger, 1937, chap. 28, p. 638.

20. Bordley, J.: Disappearance of Diabetes During the Development of Cirrhosis of the Liver, Bull. Johns Hopkins Hosp. **47**:113, 1930. Fiessinger, N.:

(Footnote continued on next page)

De Lange [21] reported a case of coexisting cirrhosis of the pancreas and the liver in a child whose first symptom was that of jaundice on the seventh day of life. The association is not difficult to understand. Diabetes mellitus is one of the commonest spontaneous diseases in man associated with fatty infiltration of the liver, and fatty infiltration has been considered a frequent precursor of cirrhosis.[22] Patek,[23] in his series of 386 cases of Laënnec's cirrhosis, found "an unexpectedly high incidence" (5 per cent) of diabetes, mentioning 13 cases of Laënnec's cirrhosis in which a diagnosis of diabetes mellitus was made and 8 other cases in which tests revealed dextrose tolerance curves characteristic of diabetes mellitus. There is no indication, however, as to whether intravenous or oral dextrose tolerance tests were used in these cases. This is a matter of utmost importance, for oral dextrose tolerance tests do not differentiate between pancreatic diabetes and hepatic disease.[24] An intravenous dextrose tolerance test has recently been described by Soskin,[1] however, that allows for this important differentiation. "One-third gram of dextrose per Kg. body weight, in a 50 per cent aqueous solution, is injected intravenously within a period of three to five minutes. Blood samples are taken before sugar administration, and at one-half, one and two hours thereafter. We used arterial blood obtained by finger puncture and the micromodification of the Somogyi-Shaffer-Hartman method for true blood sugar." This method is well suited for infants and was used in this case. The blood sugar curves of 30 normal controls, 25 patients with mild diabetes and 50 patients with early hepatic disease were determined by this method by Soskin. The blood sugar in all the normal controls returned to the preinjection level by sixty minutes. The blood sugar in the patients with hepatic disease returned to the preinjection level by one hundred and twenty minutes (25 per cent returning in sixty minutes). In none of the patients with diabetes mellitus, however, had the blood sugar level returned to the initial level after one hundred and twenty minutes;

Diabetes in the Course of Alcoholic Cirrhosis, J. de méd. et chir. prat. **99**:307, 1928. Azerad, E., and Negay, P.: Hepatic Cirrhosis in Diabetes Mellitus, Prat. méd. franç., Paris **9**:200, 1928. Bochut, L., and Volmat, H.: Diabetes with Cirrhosis, Rev. de méd., Paris **33**:753, 1913. Careddu, G.: Atrophic Cirrhosis of the Liver in Children, Clin. pediat. **12**:555, 1930.

21. de Lange, C.: Cirrhosis of the Pancreas and Liver in an Infant, Am. J. Dis. Child. **34**:372 (Sept.) 1927.

22. Chaikoff, I. L., and Connor, C. L.: Production of Cirrhosis of the Liver of the Normal Dog by High Fat Diets, Proc. Soc. Exper. Biol. & Med. **43**:638, 1940. Connor, C. L.: Etiology and Pathogenesis of Alcoholic Cirrhosis of the Liver, J. A. M. A. **112**:387 (Feb. 4) 1939.

23. Ratnoff, O. D., and Patek, A. J.: The Natural History of Laënnec's Cirrhosis, Medicine **21**:207, 1943.

24. Soskin, S., and Mirsky, I. A.: The Influence of Progressive Toxemic Liver Damage upon the Dextrose Tolerance Curve, Am. J. Physiol. **112**:649, 1935.

this fact allows for a clear, easy differentiation. This test was done in the case being reported, and pancreatic diabetes was, accordingly, excluded. The blood sugar had returned to its preinjection level before one hundred and twenty minutes. Autopsy confirmed the antemortem anatomic diagnosis, based on this test, of a normal pancreas and established hepatic cirrhosis as responsible for the disturbance of carbohydrate metabolism. This test had heretofore been performed only on adults. It is suggested that it is applicable to infants as well.

That the high blood sugar level and the mellituria represented galactosemia and galactosuria was considered, unfortunately, only shortly before the patient's death. Lactose forms a large part of ingested carbohydrate during infancy and is removed in large part from the blood as galactose almost exclusively by the liver. There is, moreover, no significant renal threshold for galactose, which is excreted when present in only small quantities in the blood. While absolute identification of the reducing substance as galactose was not made, the extremely slow rate of fermentation of the reducing substance in the urine in the face of severe hepatic dysfunction suggested that this may well have been the case. There are only 8 reports in the literature on spontaneous galactosuria,[25] and of these only 2 [25a,f] have been reported hitherto in which jaundice was a complicating factor. The clinical picture of nutritional disturbance, anemia, hepatomegaly, splenomegaly, mellituria and albuminuria was commonly noted, with prompt improvement following the institution of a milk-free (lactose-free) diet.

The relatively high nonprotein nitrogen level in our case was not due to a high amino acid nitrogen secondary to a hepatic deficiency in deamination with urea formation—a process known to occur exclusively in the liver.[26] The urea nitrogen was constantly elevated and constituted its normal 50 per cent of the total nonprotein nitrogen. This in the presence of a persistent albuminuria, hyaline and granular casts suggested concomitant renal involvement. It has long been recognized that a physiologic relationship exists between the liver and

25. (*a*) Goëppert, F.: Galaktosurie nach Melchzuckergabe bei angeborenem, familiärem, chronisichen Leberleiden, Berl. klin. Wchnschr. **54**:473, 1917. (*b*) Fanconi, G.: Spontaneous Galactosuria in a Child with Neurofibromatosis, Jahrb. f. Kinderh. **138**:1, 1933. (*c*) Reuss, A.: Spontaneous Galactosuria in a Nursling, ibid. **82**:219, 1933. (*d*) Bansi, H. W.: Paroxysmal Galactosuria, Klin. Wchnschr. **11**:21, 1932. (*e*) Mason, H. H., and Turner, M. E.: Chronic Galactemia, Am. J. Dis. Child. **50**:359 (Aug.) 1935. (*f*) Lescovar, E.: Spontaneous Galactosuria in the Infant, Ztschr. f. Kinderh. **63**:590, 1942. (*g*) Norman, F. A., and Fashena, G. J.: Chronic Hypergalactosemia, Am. J. Dis. Child. **66**:531 (Nov.) 1943. (*h*) Mellinkoff, S.; Roth, B., and MacLaggan, J.: Galactosemia with Hepatic Damage, J. Pediat. **27**:338, 1945.

26. Mann, F. C.: Effects of Complete and of Partial Removal of the Liver, Medicine **6**:419, 1927.

kidney, as the kidney is the chief means of eliminating exogenous or endogenous toxins that a damaged liver cannot neutralized or detoxify. The liver-kidney syndrome, beginning with hepatic damage and followed by parenchymatous renal degeneration, has been studied extensively by Helwig and Schutz.[27] Extensive hepatic necrosis with concomitant renal degeneration was reported also by Degener and Jaffé [28] in the case of a newborn infant, while Pytel [29] also called attention to the complementary nature of the lesions in the liver and kidney. Henrikson [30] found albumin in the urine of approximately one half of 151 patients with cirrhosis, casts in the urine of 51 and sugar in that of 10. György [31] found that necrotizing nephrosis in combination with hepatic necrosis could be produced simultaneously by purely dietary means. This is particularly interesting in view of the great stress placed on dietary deficiency as related to cirrhosis. Albuminuria and granular casts were noted in this patient early in the second week of life, weeks before ascites was noted. Renal stasis resulting from pressure of the ascitic fluid on the renal vein could, therefore, not be postulated for this patient. It is interesting to note, however, that in all the reported cases of galactosuria in infancy there was associated persistent albuminuria which disappeared when the patient was placed on a milk-free diet. Whether this was due to a directly injurious effect of excreted galactose or to tissue damage secondary to the low level of glucose in the blood observed in galactemia [25e] is still a moot question.

SUMMARY

Cirrhosis of the liver is rarely suspected prior to the onset of jaundice, ascites or hemorrhage from esophageal varices, all grave prognostic signs. In view of the good results obtained by a correction of dietary deficiencies in cirrhosis, early recognition of hepatic disease and its differentiation from extrahepatic disorders are matters of some moment. Soskin's intravenous tolerance test, readily performed and easily interpreted, detects cases of early hepatic disease manifested by a disturbance in carbohydrate metabolism and differentiates between pancreatic and hepatic diabetes.

27. Helwig, F. C., and Schutz, C. D.: A Liver-Kidney Syndrome, Surg., Gynec. & Obst. **55**:570, 1932; Further Contribution to the Liver-Kidney Syndrome, J. Clin. & Lab. Med. **21**:264, 1935.

28. Degener and Jaffé, R.: Ausgedehnte Lebernekrosen bei einem Säugling, Centralbl. f. allg. Path. u. path. Anat. **35**:556, 1924-1925.

29. Pytel, A.: Clinical Characteristics of Hepatorenal Syndrome, Wien. klin. Wchnschr. **50**:965, 1937.

30. Henrikson, E. C.: Cirrhosis of the Liver, Arch. Surg. **32**:413 (March) 1936.

31. György, P.: Experimental Hepatic Injury, Am. J. Clin. Path. **14**:67, 1944.

A case of congenital biliary cirrhosis is reported, in which melliturıa, first noted in the second week of life, was an early indication of hepatic dysfunction. The exclusion of pancreatic diabetes was possible in a 2 month old infant on the basis of the intravenous tolerance test, previously reported as done only on adults.

The probability of galactemia and galactosuria is considered.

The unusual features of congenital biliary cirrhosis without obstruction of the bile ducts and with concomitant parenchymatous degeneration of the renal tubules are discussed.

707 Fullerton Avenue.

PATHOLOGIC STUDY OF THE ACUTELY INFLAMED HUMAN PHARYNX IN INFLUENZA A INFECTION

J. M. ADAMS, M.D.

M. M. PENNOYER, M.D.
AND
A. M. WHITING, M.D.
MINNEAPOLIS

ALTHOUGH records of influenza epidemics go back to the tenth century, little is known of the pathologic changes caused by the virus of influenza in man. MacCallum [1] stated in the latest edition (seventh, 1940) of his textbook, that "no one died of influenza alone without secondary infection with bacteria," and that following extensive studies he and his associates were "entirely uninformed as to the nature of any changes in the internal organs which may result from influenza as such." The most characteristic clinicopathologic feature of the more recent proved epidemics of influenza in man is an acute pharyngitis. Francis [2] has stated (1941) that the virus of influenza exerts its initial and primary effect on the epithelium of the upper respiratory tract. Bloomfield and Harrop [3] recognized and emphasized this fact in 1919. They reported, "Early in the course of the epidemic we noticed an unusual bright red appearance to the throat in most of the patients."

The present study is concerned with the microscopic pathologic changes of the acutely inflamed human pharynx in an epidemic of proved influenza A infection. To our knowledge, no previous studies have recorded these pathologic changes in man in the uncomplicated disease. Francis and Stuart-Harris,[4] however, have described the nasal histologic changes accompanying epidemic influenza infection in the ferret. They stated in summary, "During the acute stage of infection the respiratory epithelium of the nasal mucous membrane undergoes necrosis with des-

From the Department of Pediatrics, University of Minnesota Medical School.

1. MacCallum, W. G.: A Textbook of Pathology, ed. 7, Philadelphia, W. B. Saunders Company, 1940.

2. Francis, T., Jr.: The Significance of Nasal Factors in Epidemic Influenza, in Problems and Trends in Virus Research, Philadelphia, University of Pennsylvania Press, 1941.

3. Bloomfield, A., and Harrop, G. A., Jr.: Clinical Observations on Epidemic Influenza, Bull. Johns Hopkins Hosp. **30**:1, 1919.

4. Francis, T., Jr., and Stuart-Harris, C. H.: Studies on the Nasal Histology of Epidemic Influenza Virus Infection in the Ferret: I. The Development and Repair of the Nasal Lesion, J. Exper. Med. **68**:789, 1938.

quamation of the superficial cells and exudation into the air passages, and an inflammatory reaction occurs in the submucosa." More details of the changes in the ferret will be referred to in the body of this paper as they compare with the changes in man. The histopathologic changes in mice inoculated with influenza virus have been recorded by many workers, but one of the most complete and recent reports is that of Nelson and Oliphant,[5] of the National Institute of Health. The disease as artificially produced in the mouse is primarily a pneumonic process, but the cellular changes are consistent with those observed by us in the human pharyngeal tissues and exudate. The cellular exudate in the animals consisted of about two-thirds small mononuclear cells and one-third polymorphonuclear leukocytes. These workers recorded damage to the bronchial epithelium, with peribronchial infiltration chiefly by small mononuclear cells. Straub[6] also expressed the belief that the destruction of large portions of the bronchial epithelium is the most conspicuous feature in mice dying from influenza.

METHOD OF STUDY

In our study, swabbings of the pharynx were made in varying stages of the disease in an epidemic of proved influenza A infection. A dry cotton swab on a long applicator was thoroughly rubbed over the posterior and lateral pharyngeal walls, and the material obtained thereby was immediately transferred to a glass slide which had previously been prepared with Mayer's solution (egg albumin and glycerin) as a fixative. Without delay, the slides were stained with a modified hematoxylin and eosin stain, and cover slips were applied. Over 300 slides were made with material from the pharynxes of young adults and children suffering with acute influenza A infection. The causative virus of this epidemic was identified by inoculating throat washings intra-allantoically into the developing chick embryo, as well as by demonstrating a significant increase of specific neutralizing antibodies in the patient's serum, as measured by the Hirst hemagglutinin inhibition test.[7] Control pharyngeal smears were made in a similar manner from young adults and children ostensibly well at the time of the epidemic, and smears were also made six months later, when there were no known cases of influenza in the community.

The amount of tissue obtained by this method varied somewhat from patient to patient, but it was found that by gently rolling the applicator over the slide more material was left on the slide than when the applicator was smeared over the surface. However, the variability of the material on the slides precluded an exact quantitative evaluation of the specimens as a whole.

PATHOLOGIC OBSERVATIONS

The pharynx appeared beefy red in the acute stage of the disease, and many of the patients complained of sore throat. Hyperemia was

5. Nelson, A. A., and Oliphant, J. W.: Histopathological Changes in Mice Inoculated with Influenza Virus, Pub. Health Rep. **54**:2044, 1939.

6. Straub, M.: The Microscopical Changes in the Lungs of Mice Infected with Influenza Virus, J. Path. & Bact. **45**:75, 1937.

7. Hirst, G. K.: The Quantitative Determination of Influenza Virus and Antibodies by Means of Red Cell Agglutination, J. Exper. Med. **75**:49, 1942.

a rather common condition, and often the swabbings elicited bleeding of a mild character. Material from the pharynx was readily obtained in most instances.

The low power inspection of the slides revealed large numbers of sloughing epithelial cells. This finding is striking in contrast to the purulent leukocytic type of exudate most frequently observed in the ordinary case of pharyngitis or in infections of the upper and lower respiratory tract of bacterial origin. We frequently saw large sheets of epithelial cells; this may be an index of the destruction of this tissue by the virus. Dividing nuclei were observed, indicating proliferation of epithelium.

A mononuclear exudate stood out as the most singular pathologic change in the human pharyngeal exudate. This type of cellular response is quite consistent with the pathologic changes recorded in cases of other types of virus infections and of influenza itself in lower animals. On microscopic examinations of the lungs of mice and ferrets, McIntosh and Selbie [8] found a great increase of mononuclear cells in the alveolar walls. Francis and Stuart-Harris,[4] however, using "relatively large doses" of epidemic influenza virus in the ferret, recorded a high degree of destruction of the epithelium of the respiratory tract and "a rich exudate chiefly polymorphonuclear in type in the air passages." McCordock and Muckenfuss,[9] using vaccinia virus in experimental animals, found the reaction to vary with the quantity of virus used. The concentrated dose produced an acute hemorrhagic type of histologic response with necrosis, whereas the more dilute dose produced a proliferative response with mononuclear cellular infiltration. Francis and Stuart-Harris [4] have called attention to mononuclear infiltrations during the stages of repair and regeneration in ferret epithelium. Rivers [10] has pointed out that acute inflammations occur in many virus diseases and, if secondary infections do not intervene, the inflammatory process is usually characterized by an infiltration of mononuclear cells.

In our experience the mononuclear exudate, made up mostly of small lymphocytes, histiocytes and plasma cells, varied in amount and was often mixed with a few polymorphonuclear cells, many of which were degenerating, with pyknotic nuclei. Early in the course of the infection,

8. McIntosh, J., and Selbie, F. R.: The Pathogenicity to Animals of Viruses Isolated from Cases of Human Influenza, Brit. J. Exper. Path. **18**:334, 1937.

9. McCordock, H. A., and Muckenfuss, R. S.: The Similarity of Virus Pneumonia in Animals to Epidemic Influenza and Interstitial Bronchopneumonia in Man, Am. J. Path. **9**:221, 1933.

10. Rivers, T. M.: Some General Aspects of Pathological Conditions Caused by Filterable Viruses, Am. J. Path. **4**:91, 1928.

the mononuclear exudate frequently represented the predominant cellular response. These reactions are shown in the accompanying figure (fig. 1).

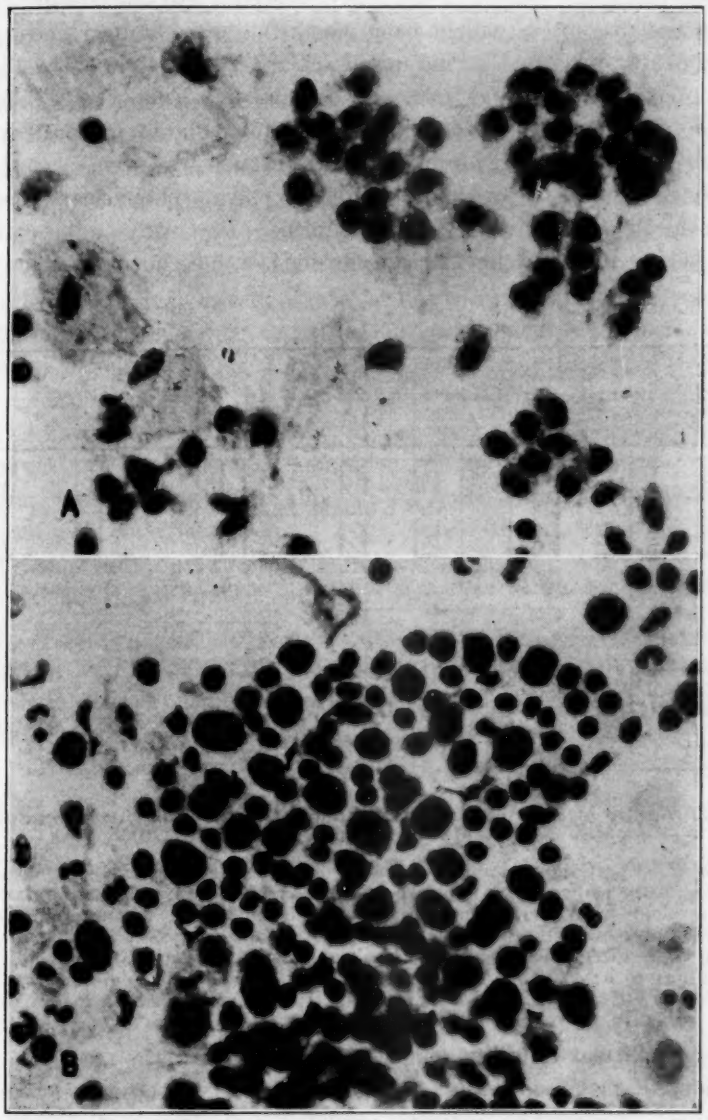

Fig. 1.—*A*, photomicrograph of mononuclear exudate from the pharynx of a patient with influenza A infection; *B*, photomicrograph showing a mononuclear exudate from a pharyngeal smear of a patient with influenza A infection. Hematoxylin and eosin stain.

In order to determine the quantitative leukocytic reactions, smears from 35 patients with definite serologic responses were studied in detail.

Twenty-four (68.5 per cent) of the patients had a predominant mononuclear exudate as illustrated in figure 2. No leukocytes could be found in 8 of the specimens, and 3 others showed a predominantly polymorphonuclear reaction. Only 1 of the latter showed a marked change. It is of interest that in the patient from whom this was obtained pneumonia subsequently developed. Pharyngeal smears from a series of well persons, prepared in May and June, were examined for leukocytes. Although 17 per cent of these were found to have a slight to moderate polymorphonuclear exudate, no mononuclear reaction was seen.

An exudate made up almost entirely of polymorphonuclear cells was found in 18 per cent of the total specimens. For many of the patients from whom these specimens were obtained, a subsequent complication,

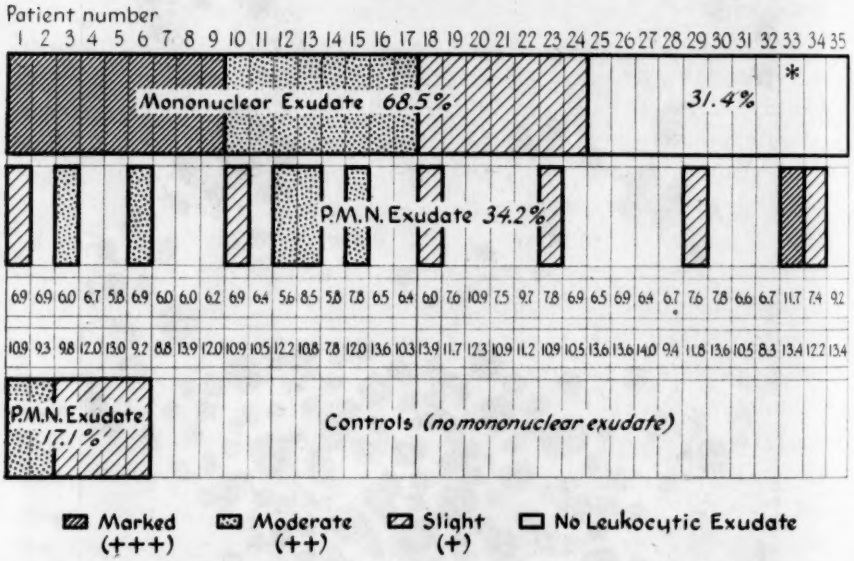

Fig. 2.—Chart showing the relationship of mononuclear and polymorphonuclear exudate in the patients with influenza A infection, compared with those of controls. The serologic responses of the patients are also indicated, the upper column of figures representing the titer in the acute phase and the lower column, the titer in the convalescent phase of each individual patient. The titers are expressed in logarithms to base 2, i. e., a rise from 6 to 10 equals a sixteenfold increase, or 1:64 to 1:1,024. The asterisk indicates that pneumonia developed in this patient.

such as follicular tonsillitis, sinusitis or pneumonia, was recorded. We considered this of interest as a possible index to secondary bacterial invasion. Twenty-two patients exhibited secondary bacterial complications (otitis media, follicular tonsillitis, scarlet fever or pneumonia). Of these, a mononuclear exudate was recorded for 18 per cent in the early stages of the disease, whereas 82 per cent had a predominant polymorphonuclear response at the same period. Fifty-five per cent of the slides were prepared during the first two days of the patients' illness.

COMMENT

A study of cells from the pharynx of the living patient is quite easily carried out by means of the technic outlined. The interpretation of data so obtained is not without a chance of error, as it is impossible to diagnose specific disease processes by this simple procedure alone. It is likewise impossible to determine the particular cells which are harboring the influenza virus. However, with the almost constant finding of degenerated epithelium in pharyngeal smears in influenza, it would appear that in the human subject the virus attacks squamous epithelium of the pharynx as well as the columnar and cuboidal epithelium of the respiratory tract. It seems possible that certain significant deductions may be made from this type of study. The predominantly bacterial and fungal infections are readily diagnosed from a study of swabbings from the throat on the basis of the exudate and other specific organisms such as bacteria and fungi, for example, Monilia albicans. Frisch and associates [11] have been able to differentiate between the mildly, moderately and critically ill pneumonia patients by means of pneumococcus counts on the admission sputum. The observations for patients with uncomplicated influenza are quite different from those for patients with bacterial infections, a fact which leads us to conclude that the primary difference probably is in the type of infection, whether it be bacterial, fungal or viral in nature.

As first pointed out by McCordock and Muckenfuss,[9] the pulmonary lesions of various virus diseases are extremely similar. They stated that three distinct elements can be recognized in the lesion that MacCallum has designated interstitial bronchopneumonia. Two of these, the cellular infiltration of the interstitial tissue and the peribronchial exudate composed of large mononuclear cells, dense masses of fibrin and red cells, are regarded as a reaction to injury by the virus. Both of these changes can be produced experimentally with virus alone, and they are found constantly in persons dying of a virus disease, such as measles, in regions of the lung where bacteria cannot be demonstrated. The third element is the result of a secondary bacterial infection that calls forth a fibropurulent exudate in the bronchioles and connecting alveoli. Golden,[12] who has recently reported the pathologic changes in army personnel dying from "atypical pneumonia," pointed out that the characteristic lesion in this condition is comparable with that in

11. Frisch, A. W.; Price, A. E., and Myers, G. B.: Pneumococcic Pneumonia: Prognostic Significance of Number of Pneumococci in Sputum in Relation to Therapy, Bacteremia, Type, Leukocyte Count, Duration of Disease, Age, and Degree of Involvement, J. Clin. Investigation **22**:207, 1943.

12. Golden, A.: Pathologic Anatomy of "Atypical Pneumonia, Etiology Undetermined": Acute Interstitial Pneumonitis, Arch. Path. **38**:187 (Oct.) 1944.

uncomplicated influenzal or measles pneumonia. His review of the literature on the pathology of atypical pneumonia reveals the same characteristic lesion. McNaught,[13] in a review of the pathology of virus pneumonia, stated, "The pathologic changes in the lungs of the few cases coming to autopsy are quite similar to those of known virus etiology and are characterized by alveolar exudate consisting largely of mononuclear cells, with polymorphonuclears conspicuously rare." It is important here to call attention to the recent significant investigations of the Commission on Acute Respiratory Diseases,[14] who succeded in transmitting the causative agent of primary atypical pneumonia to two successive groups of well persons by the use of bacteria-free filtrates

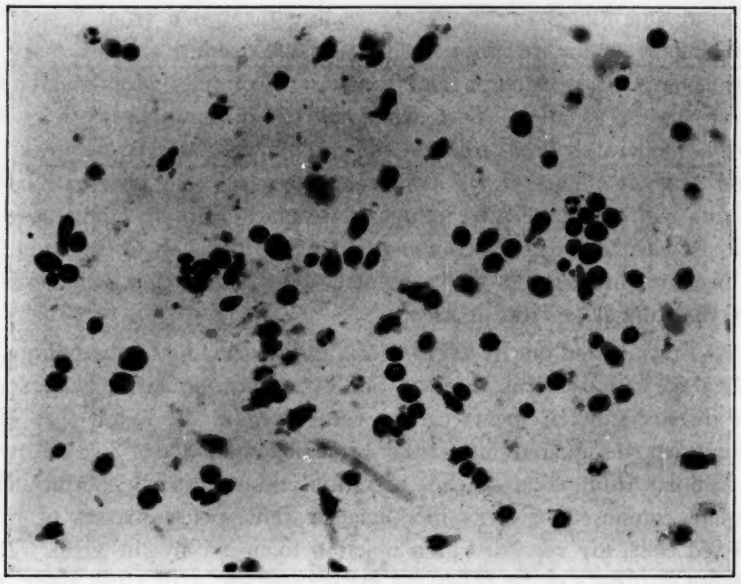

Fig. 3.—Photomicrograph of the pharyngeal exudate from a patient with virus pneumonia to show a predominantly mononuclear exudate. There are a few scattered polymorphonuclear cells in the preparation. Hematoxylin and eosin stain.

from patients with "atypical pneumonia, etiology undetermined." It now seems quite proper, therefore, to speak of this disease as "virus pneumonia" or "virus pneumonitis."

Similarly, reactions of the pharyngeal tissues as a result of infections by pneumotropic viruses appear within the limits of the method to be

13. McNaught, J. B.: Virus Pneumonia: A Review of the Pathology, California & West. Med. **59**:220, 1943.

14. Transmission of Primary Atypical Pneumonia to Human Volunteers, Commission on Acute Respiratory Diseases, Fort Bragg, N. C., J. A. M. A. **127**:146 (Jan. 20) 1945.

uniform and distinct from other types of infection. A review of the slides prepared in a like manner from patients studied by one of us (J. M. A.) in an epidemic of infant virus pneumonitis in 1940 and 1941 [15] reveals some observations similar to those reported in this paper, particularly to those concerning the epithelial cells. The specimens revealed sloughing of the epithelial cells in large sheets. There was little leukocytic exudate in the pharyngeal material from the infants; however 85 per cent of the specimens were positive for cytoplasmic inclusion bodies and over 50 per cent were classified as heavily positive. This observation correlated directly with finding of inclusion bodies seen in the bronchial, bronchiolar and alveolar epithelium of the patients dying from the disease. The pathologic picture in every other way corresponds with that of the acute interstitial pneumonitis reported in the literature. Pinkerton, Smiley and Anderson,[16] in a recent report on giant cell pneumonia, compared their observations with those reported by us and stated that the cytoplasmic inclusions were relatively small and constant in size and shape, in contrast to the pleomorphism of the inclusions seen in giant cell pneumonia and in distemper. "The absence of discoverable nuclear inclusions in carefully studied material from many cases also suggests that this is probably a different type of virus pneumonitis." [16]

More recent studies by us of pharyngeal exudate from adults with virus pneumonia reveal a predominantly mononuclear response in many of the patients studied. A photomicrograph of a pharyngeal smear from a patient with virus pneumonia is shown in figure 3, illustrating the mononuclear exudate.

SUMMARY AND CONCLUSIONS

1. The most characteristic clinicopathologic feature of epidemic influenza, as seen in a recent proved epidemic, was acute pharyngitis.

2. The pharyngeal smears of over 300 young adults and children suffering from influenza A infection were studied, as well as similar tissues from control groups, both with and without "common colds," during and six months after the epidemic. Increased destruction of pharyngeal epithelial tissues was definite, as compared with the control specimens.

3. The single most important micropathologic feature of the pharyngeal exudate of these patients with influenza was a mononuclear

15. Adams, J. M.; Green, R. G.; Evans, C. A., and Beach, N.: Primary Virus Pneumonitis: A Comparative Study of Two Epidemics, J. Pediat. **20**:405, 1942.

16. Pinkerton, H.; Smiley, W. L., and Anderson, W. A. D.: Giant Cell Pneumonia with Inclusions, Am. J. Path. **21**:1, 1945.

exudate, which is consistent with the pathologic changes in certain other types of virus infections, both in man and in animals.

4. In our study a polymorphonuclear exudate frequently presaged a complicating secondary bacterial infection.

5. A study of pharyngeal smears is helpful in the differential diagnosis of bacterial, fungal and viral diseases involving the human throat.

Case Reports

HISTOPLASMOSIS IN INFANCY

HERBERT V. DAVIS, M.D., and FRANK C. NEFF, M.D.
KANSAS CITY, KAN.

WE WISH to add 2 cases of histoplasmosis in infancy to the gradually enlarging list of cases of this disease in children and adults being reported. All writers mention its discovery by Darling in 1905, while he was doing special research work with kala-azar in Panama. A recent valuable and extensive report with a review is that of Parsons and Zarafonetis.[1] At first thought to be a rare tropical disease, histoplasmosis is now showing evidence of a wide spread, at least in the United States and other temperate climates. Furthermore, it has some symptoms which belong to certain other diseases of childhood from which it must be differentiated, among them, leukemia and tuberculosis. In view of the present limited knowledge of the method of spread, the disease assumes importance in the field of public health and is becoming a matter of concern to workers there.

The infection of histoplasmosis is manifest in many different ways. It may be localized, as in the production of an ulcer of the pharynx; it may invade one organ, or it may be generalized, involving most of the viscera. It attacks the reticuloendothelial system, and its course is characterized by chronicity, irregular pyrexia, hepatomegaly, splenomegaly, emaciation, anemia and leukopenia. Its small encapsulated organism resembles the Leishman-Donovan bodies of kala-azar and is found in the cells of the liver, foam cells of the bone marrow and elsewhere.

As regards the incidence, it is reported to be found at any age, but in as many as 25 per cent of the cases the disease occurs in children, usually under 6 months. Its manifestations in children differ somewhat from those in adults in that the course is much shorter and the evidence more extensive. The average duration of the disease has been reported

From the Department of Pediatrics, University of Kansas School of Medicine.

We were assisted in this study of the tissues and the fungus by George Walker, Department of Pathology, and Clarence Weber, Head of Clinical Laboratory Medicine.

1. Parsons, R. J., and Zarafonetis, C. J. D.: Histoplasmosis in Man: Report of Seven Cases and Review of Seventy-One Cases, Arch. Int. Med. **75**:1 (Jan.) 1945.

as from three weeks to eight months. The opinion seems to be developing that the disease is more common in the Caucasian race, in males and among the rural population.

The organism, Histoplasma capsulatum, is regarded thus far as not having been found in nature, but it may be transmitted in animals. The mode of infection, the period of incubation and the epidemiology are unknown.

The organisms will grow on most of the usual mediums and require from six to thirty days. The mycelium at first forms an indurated colony assuming the color of the medium; later, it becomes a white cot-

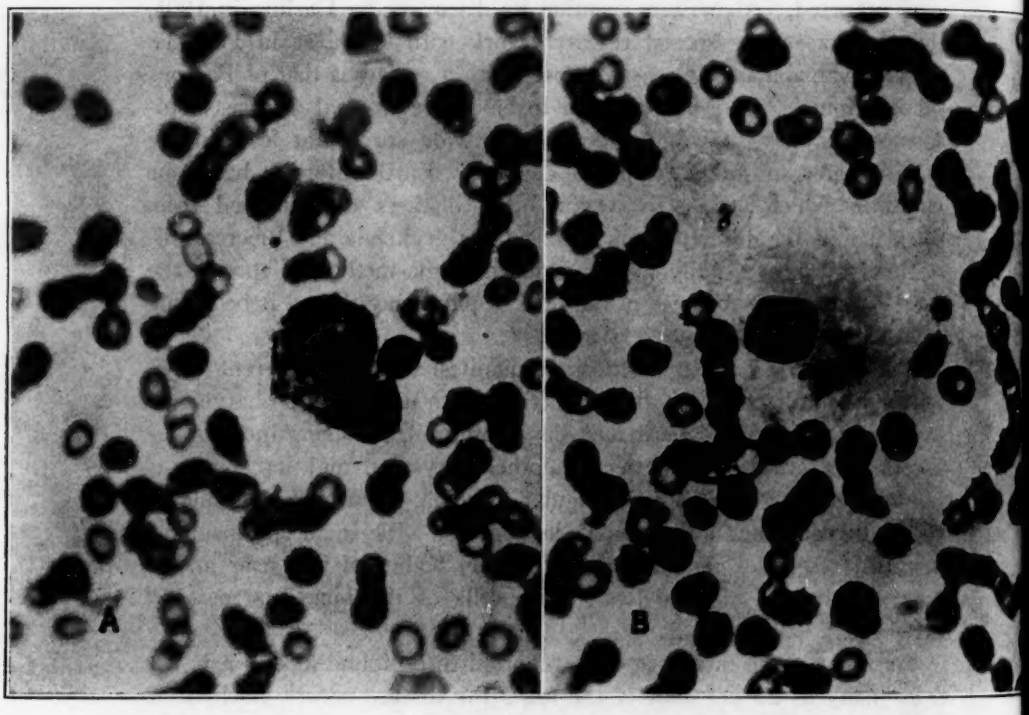

Fig. 1 (case 2).—*A*, a large mononuclear phagocyte containing six organisms and one ingested leukocyte; *B*, a large mononuclear cell containing twelve organisms.

tony growth which with age changes its color to buff or brown. The yeast bodies can be cultured by incubating the mycelium at 37 C. in 0.05 per cent solution of sodium hydroxide, after which it is transferred to sealed blood agar plates at 37 C. The colonies grossly resemble colonies of Staphylococcus aureus.

H. capsulatum exists in two forms. As seen in the tissues, it is an oval yeastlike body 1 to 5 microns in diameter, with a sharply defined, clear, achromatic capsule with a central dark-staining chromatin mass usually possessing a round vacuole (fig. 1). On culture it may also

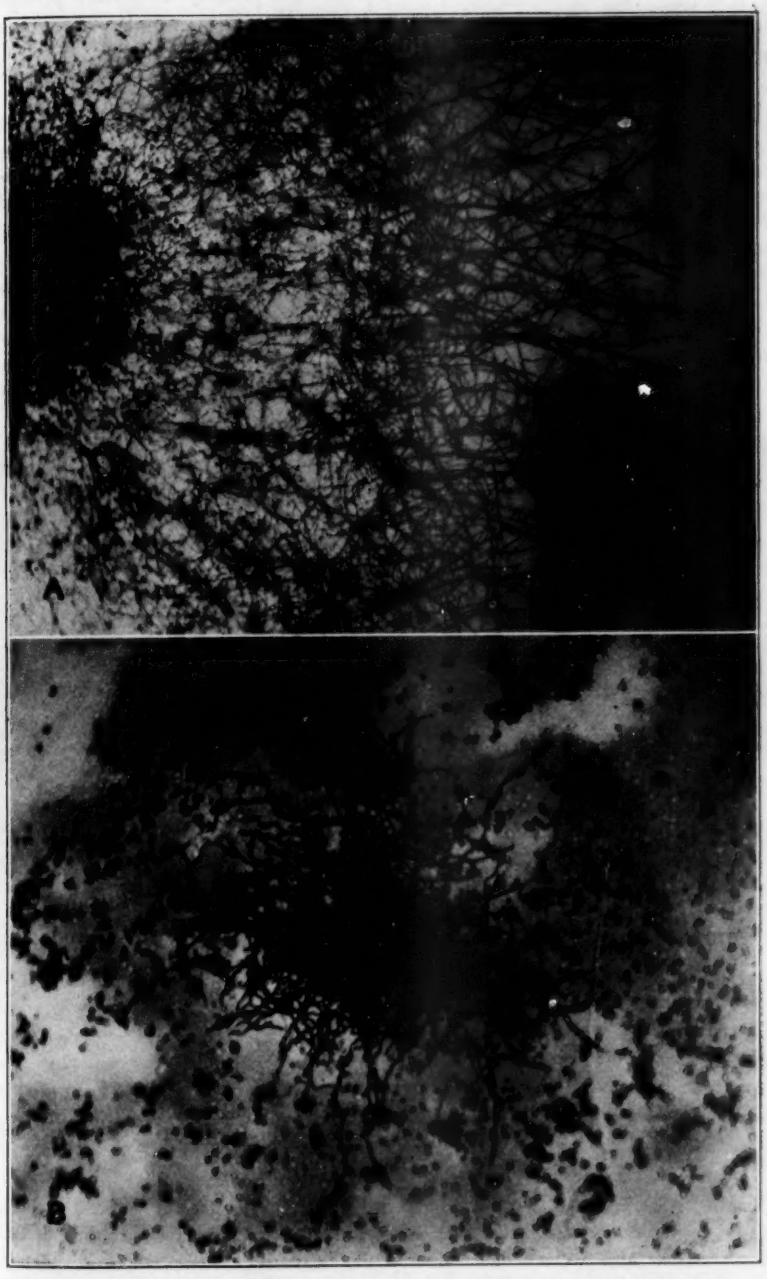

Fig. 2 (case 2).—*A*, mycelial form (Sabouraud's medium; × 400); *B*, mycelial form (outgrowth on Sabouraud's medium; low power, × 160).

occur as a yeast form but more often is a white cottony mycelium possessing microscopic septate hyphae or filaments bearing small round pyriform chylomydospores (fig. 2).

Our 2 cases exemplify the disease as seen in infancy.

REPORT OF CASES

CASE 1.—C. M., a white girl aged 9 months, entered the University of Kansas Children's Hospital Nov. 11, 1944, from Pilot Grove, Mo. There was a history

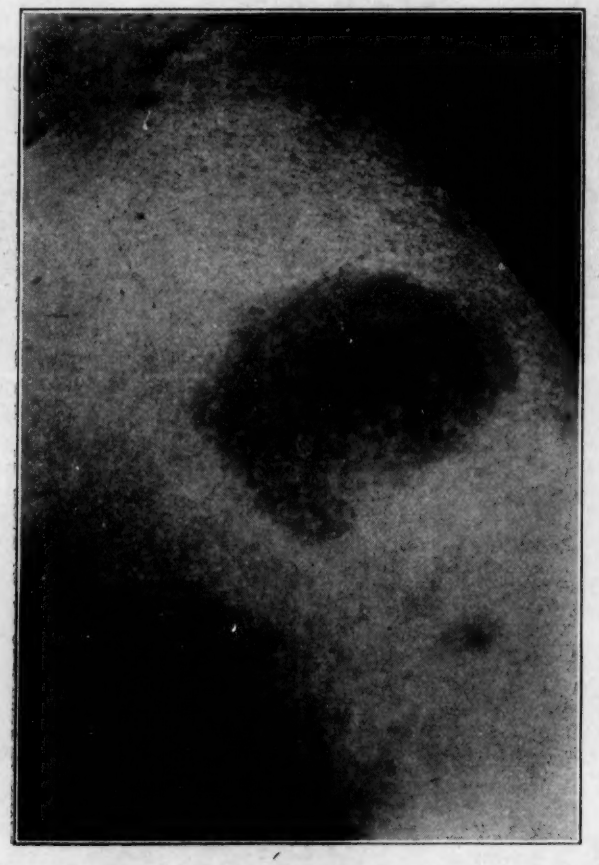

Fig. 3 (case 2).—A positive reaction to a cutaneous test made on the patient's mother.

of having been perfectly well four weeks prior to admission. The little girl then became irritable on being handled; she was found to have lost weight, to have become anemic and to have had a definitely progressive downhill course. Emaciation and coma developed. The child had been sent to us with a diagnosis of leukemia. At the time of admission to the hospital, she had a temperature of 104 F. The anterior fontanel was sunken, and the conjunctivas and mucous membranes were extremely pale. There were numerous sonorous rales throughout the

chest and a soft systolic murmur over the precordium. The abdomen was prominent and the liver firm and palpable 4 fingerbreadths below the costal margin; the spleen was palpable down to the left anterior superior iliac spine. There was pitting of the lower extremities from edema.

On immediate examination, the blood smear showed 2,840,000 erythrocytes per cubic millimeter; the hemoglobin was 7 Gm. per hundred cubic centimeters, or 45 per cent; there were 16,380 white corpuscles per cubic millimeter, with 73 per cent lymphocytes (many immature forms) and 24 per cent polymorphonuclear leukocytes. No further opportunity for making laboratory studies was possible because of the death of the child one hour after admission. The laboratory observations of the referring physician revealed negative results from culture of the blood, leukopenia (the white corpuscles ranging from 1,250 to 3,200 per cubic millimeter) and presence of anemia. However, there had been normal blood chemistry and normal results from serologic tests, urinalyses and examination of the spinal fluid. The patient had been sent to us with a diagnosis of aleukemic lukemia, which was similarly our impression at the time of death. Microscopic studies of

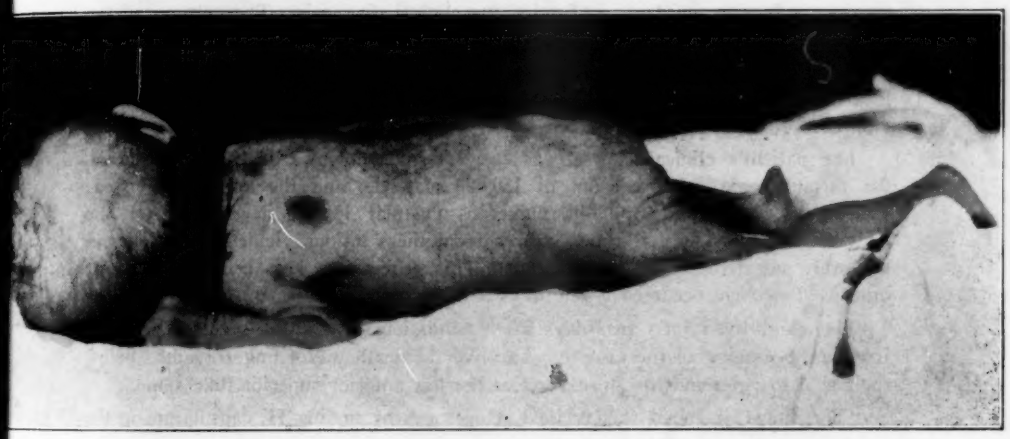

Fig. 4.—Terminal pustular cutaneous lesions (did not contain H. capsulatum).

materials obtained at autopsy were made, and H. capsulatum was found in the spleen, liver, myocardium, adrenal glands, lymph nodes, kidneys and lungs.

Case 2.—H. M., a white boy aged 5 months, entered the University of Kansas Children's Hospital from Excelsior Springs, Mo., with the history of unexplained fever and cough. The child had been normal at term and delivery, having been perfectly well until one month prior to admission; he was sent in because of an infection of the upper respiratory tract. There had been three exacerbations and remissions. We found evidence of infection of the upper respiratory tract, with numerous moist rales and distention of the abdomen. There was no enlargement of the spleen and liver at that time. Cough was present, and roentgen visualization showed the presence of bronchitis.

The blood count gave the following results: 2,950,000 red blood cells per cubic millimeter, with a hemoglobin level of 51 per cent, and 4,100 white corpuscles, with a normal differential count; the blood platelets numbered 150,000. There was nothing abnormal in the sedimentation rate, in the chemistry of the blood and the spinal fluid or in the results of the urinalyses.

During the early period of hospitalization, the treatment consisted of the administration of sulfadiazine and penicillin, and the giving of small transfusions of whole blood and supportive therapy, all of which were of no avail. The fever (range of temperature 99 to 103 F.), the leukopenia (with a white cell count ranging from 2,100 to 4,100) and the anemia persisted. Loss of weight and enlargement of the spleen and liver appeared on the twelfth hospital day. At that time the clinical diagnosis of histoplasmosis was regarded as probable. Aleukemic leukemia, Gaucher's disease and brucellosis were ruled out. Studies of the sternal marrow revealed from 1 to 17 yeastlike bodies (fig. 1) in the cytoplasm of the monocytes and foam cells. A biopsy of an inguinal lymph node showed acute and chronic adenitis but no specific evidence of histoplasmosis. Routine cultures of the blood done at admission gave negative results ten days later and were discarded. After histoplasmosis was considered clinically, cultures of the blood, sternal marrow and the stool and of specimens taken at biopsy were started on Sabouraud's medium, dextrose blood agar, tryptose-phosphate broth with 1 per cent dextrose added and brain-heart infusion. H. capsulatum was recovered from the blood on these mediums in from nine to nineteen days and from the sternal marrow in four to ten days, confirming the clinical diagnosis. The cultures of the stool and specimens taken at biopsy were negative.

A cutaneous test, with 0.1 and 0.2 cc. of specific skin-testing material, resulted in a negative reaction in the patient but a strongly positive reaction in the mother (fig. 3).

The patient's clinical course was progressively downward. The leukopenia, the thrombopenia, with a count of 150,000 platelets, and the anemia persisted; cachexia developed. Terminally, numerous pustular lesions developed over the body (fig. 4), and at that time cultures from these lesions yielded Staph. aureus but were negative for H. capsulatum. This sequel has also been observed by others. Petechiae occurred a few days before death.

The child lived forty-two days after admission, a total of seventy-two days from the beginning of the disease. The liver at death was 4 fingerbreadths below the costal margin, and the spleen was at the left anterior superior iliac spine.

The clinical diagnosis was verified at necropsy by finding H. capsulatum in the liver, spleen and adrenal glands.

COMMENT

At the time of writing, there had been 59 cases of histoplasmosis reported, 15 cases reviewed but not reported and our 2 cases, making a total of 76. Since 1936 the incidence has suddenly increased, as there had been only 12 cases reported until that time. It is felt that this increase may not be due alone to better recognition of the disease from the rechecking of autopsy and laboratory reports, but it is more probable that the incidence is actually on the increase. Missouri, Michigan and Tennessee appeared to have the highest incidence, Missouri having had a total of 12 cases.

Out of the 76 cases reviewed, H. capsulatum was found on examination of the blood in 4 instances; sternal marrow was studied in 9 cases and found to be positive for the organism in 5 instances. In 29 cases it has been possible to establish a diagnosis by biopsy. Cultures positive

for H. capsulatum have been obtained in 30 cases—in 10 instances from the blood, in 2 from sternal marrow, and in 18 from material taken at biopsy or autopsy.

There is no specific treatment. Stibamine glucoside (Neostam) and other antimony preparations are said to have caused a short remission. Penicillin and sulfonamide compounds are not effective. Of temporary value are whole blood transfusions.

Obituaries

ROWLAND GODFREY FREEMAN, M.D.
1859-1945

Dr. Rowland Godfrey Freeman, one of the few remaining early members of the American Pediatric Society, died in New York Nov. 14, 1945, in his eighty-seventh year.

Entering Columbia from Lawrenceville School, he received the degree of A.B. in 1883 and of M.D. from the College of Physicians and Surgeons in the City of New York in 1886. After serving as an intern at Bellevue Hospital, he studied for eighteen months in Berlin, Vienna and Paris, devoting most of his time to bacteriology and pathology. On his return to New York he was pathologist at Babies Hospital for a time.

At various times he was attending physician at St. Mary's Free Hospital for Children, New York Foundling Hospital, Nursery and Child's Hospital and New York Infant Asylum and pediatrician to Roosevelt Hospital, resigning from the latter position in 1924 and becoming consultant, at the age of 65. He was professor of pediatrics at University and Bellevue Hospital Medical College and was subsequently emeritus professor.

In the nineties, Dr. Freeman invented a practical simple home pasteurizer, which was widely used before pasteurization of milk was generally adopted. He was always deeply interested in improvement of the milk supply and was a member of the first Certified Milk Commission of the Medical Society of the County of New York. He was its president at the time of his death. He was also a valued and practical member of the Milk Commission of the Walker Gordon Laboratories.

A great believer in fresh air, he did much to improve the sanitation of the sick room, in his early days always kept at a high temperature. At Roosevelt Hospital he had an outdoor ward for patients with pneumonia.

Dr. Freeman was elected to the American Pediatric Society in 1895, when 36 years of age, and was second vice president in 1907, a member of the council from 1907 to 1914 and president in 1916. He was a very regular attendant at its meetings, and did not become an emeritus member until 1932.

Abstracts from Current Literature

Newborn

PROBLEMS ON THE CARE OF THE NEWBORN. T. MCENERY, Illinois M. J. **87**:237 (May) 1945.

Care of the newborn to be successful should depend on one who has had special training in this field and who is able to instruct those under her in the importance of little things which mean so much in the management of a nursery for the newborn. Adequate equipment of the nursery is essential. The management of the department should be under direct medical supervision.

Any infant whose weight is under 5 pounds (2,270 Gm.) should be treated as a premature infant. Breast milk is the food of choice. Feeding of vitamins and iron should be started early.

The infant's skin should be protected from excessive irritation. The practice of no bathing seems to accomplish this and is a boon to the nursing situation.

The clothing of the newborn infant should be light in weight, easy to put on and take off and not binding. The baby should be kept dry and cool.

When all of the obstacles to successful breast feeding are known, a little more diligence on the part of the obstetrician in his antepartum instruction to the mother, with the holding back of the prelacteal feedings except water, and the making of a concentrated effort to stimulate in the mother a willingness to nurse the baby, should prove helpful.

Circumcision should not be performed routinely. This operation is often done in a poor fashion. Enough of the foreskin should be removed so that after healing the skin covers the corona of the glans. Too much surgical scrubbing of the penis, abdomen and groin produces too great irritation of the skin and usually results in a pustular dermatitis. Dressing with petrolatum also leads to rashes. A piece of gauze soaked in tincture of benzoin makes for good hemostasis, and the dressing does not have to be changed.

GONORRHEAL OPHTHALMIA NEONATORUM. ALBERT A. BARAFF, Illinois M. J. **87**: 249 (May) 1945.

A case of gonorrheal ophthalmia neonatorum (infectious acute gonococcic conjunctivitis) is reported, in which blindness resulted. In violation of the laws of the State of Illinois, the case was not reported to the local health authorities, nor were the proper therapeutic measures instituted.

BARBOUR, Peoria, Ill.

EXFOLIATIVE DERMATITIS (RITTER'S DISEASE). RAUL ORTEGA and ADRIANA CASTRO, Rev. chilena de pediat. **15**:981 (Dec.) 1944.

Observations were made concerning 21 cases of exfoliative dermatitis (Ritter's disease). In 80 per cent of these, lesions appeared before the tenth day, usually about the mouth, and spread rapidly. The mortality was 60 per cent—86.7 per cent among the infants born in the maternity hospital and 25 per cent among those born at home. Prognosis in individual cases was difficult. Three of the infants were premature.

In addition to bacterial causes, contributory allergic factors and possibilities are discussed. Sulfonamide therapy was disappointing, and the authors predict better results with penicillin.

PLATOU, New Orleans, La.

Acute Contagious Diseases

PERTUSSIS SKIN TEST IN NEW BORN AND LATER INFANCY. M. KUNSTLER, Canad. M. A. J. **52**:62 (Jan.) 1945.

A group of 680 children 1 week to 8 months of age were given cutaneous tests with pertussis endotoxin. The following positive reactions were obtained: newborn, 21 per cent; 2 to 3 months, 41 per cent; 4 to 5 months, 52 per cent, and 6 to 8 months, 65 per cent. The curve plotted for the cutaneous endotoxin test with pertussis almost parallels the curve for the Schick test.

The author believes that the pertussis cutaneous test offers a reliable method of determining the state of immunity to pertussis.

MOORE, Omaha.

SUSCEPTIBILITY OF MUSKRATS AND OTHER RODENTS TO POLIOMYELITIS VIRUS (LANSING STRAIN). F. B. GORDON, J. Infect. Dis. **76**:155 (March-April) 1945.

The infection of muskrats (Ondatra zibethicus) by intracerebral inoculation of mouse-adapted poliomyelitis virus (Lansing strain) is described. The virus was transmitted through four passages in muskrats and produced a disease clinically and pathologically similar to that of other rodents infected with this virus.

White-footed mice (Peromyscus leucopus) were found to be irregularly susceptible to this virus, and ground squirrels (Citellus tridecemlineatus and Citellus richardsonii) were entirely refractory.

AUTHOR'S SUMMARY.

ACUTE POLIOMYELITIS AND ACUTE INFECTIOUS LYMPHOCYTOSIS—THEIR APPARENT SIMULTANEOUS OCCURRENCE IN A SUMMER CAMP. JEROME S. BELOFF and KENNETH M. GANG, J. Pediat. **26**:586 (June) 1945.

This report deals with an unusual group of cases related in time and space and possibly in causation. Within a period of fourteen days, 4 children, closely associated at a camp, had suggestively related illnesses. The first child had a fatal attack of bulbar poliomyelitis. The second patient became ill one week later, the clinical picture resembling that of acute infectious lymphocytosis, the disease entity recently described by Carl Smith. However, had it not been for the white blood cell count of 50,000 with 90 per cent normal lymphocytes, the diagnosis of nonparalytic poliomyelitis could have been made. At approximately the same time, another camper had a clinical picture similar to that of the second with respect to the unusual blood count and with the associated manifestations of multiple paralyses typical of poliomyelitis. The fourth patient had all the clinical and laboratory criteria of acute nonparalytic poliomyelitis except for definite tenderness of the parotid glands, which suggested the possibility of a mumps meningoencephalitis.

The differential diagnosis in these cases included acute poliomyelitis, lymphocytic choriomeningitis, mumps meningoencephalitis, tuberculous meningitis, infectious mononucleosis and acute infectious lymphocytosis. The laboratory data most strongly suggested the presence of acute poliomyelitis and acute infectious lymphocytosis as coexisting entities in these 4 cases. From the laboratory data and clinical course, 2 of the children had poliomyelitis, whereas the other patients apparently had unusual types of acute infectious lymphocytosis.

A review of the subject of infectious lymphocytosis with its differential diagnosis is presented. The possible significance of the association in one epidemic area of poliomyelitis and acute infectious lymphocytosis is discussed. It is suggested that infectious lymphocytosis might possibly represent an atypical clinical form of poliomyelitis on the basis of different strains of the poliomyelitis virus.

AUTHORS' ABSTRACT.

STUDIES ON SUSCEPTIBILITY TO POLIOMYELITIS. CHARLES ARMSTRONG and DORLAND J. DAVIS, Pub. Health Rep. **60**:710 (June 22) 1945.

Serums from 42 mothers of victims of paralytic poliomyelitis strongly neutralized the virus in 39 instances and partially neutralized it in 3; while the serums from 27 fathers strongly neutralized the virus in 23 instances, partially neutralized it in 3 and did not neutralize it in 1. These results give no indication that paralytic poliomyelitis is more apt to occur among persons whose parents do not readily produce circulating antibodies against the virus.

LESLIE, Evanston, Ill.

SMALLPOX VACCINATION AND SMALLPOX. A. ECKSTEIN, Ann. pædiat. **163**:158 (Aug.-Sept.) 1944.

The author reports 2 cases in which children vaccinated for the first time had smallpox, in spite of successful and strong vaccination reactions and fever-free intervals. During the interval there was a catarrhal prodromal stage. The course of the pox was mild.

This confirms the observed (chiefly in persons vaccinated two or more times) failure of an absolute protection by means of vaccination, when vaccination is made at the beginning of the incubation period, even though there was a strong local reaction.

These observations are of no importance in the estimation of the fundamental protection assured by vaccination; they point only to the fact that the protection does not arise so early in all circumstances as is theoretically supposed. Vaccination against smallpox at the commencement of the incubation period is no guarantee that a mild form of pox will not appear in a few days in spite of a definite vaccination reaction.

In practice this shows that persons vaccinated during the incubation of smallpox, even though the reaction is positive, should be held in quarantine for two or three weeks in order to prevent the spread of the disease.

AUTHOR'S ABSTRACT.

Acute Infectious Diseases

PENICILLIN TREATMENT OF SULFONAMIDE-RESISTANT GONORRHEA: RESULTS OF 500 CASES TREATED WITH 50,000 UNITS OF PENICILLIN. ALLEN I. JOSEY and FORREST E. KIRSHMAN, Ann. Int. Med. **22**:807 (June) 1945.

Of 500 consecutive patients with sulfonamide-resistant gonorrhea, 438, or 87.6 per cent, were apparently cured after the administration of 50,000 units of penicillin given in doses of 10,000 units every three hours over a period of five days. Forty-five of the 62 patients for whom this dosage resulted in failure responded satisfactorily to 100,000 units, making a total of 96.6 per cent of satisfactory results.

READING, Galveston, Texas.

RHEUMATIC FEVER AS A PUBLIC HEALTH PROBLEM. JEROME J. SIEVERS, Illinois M. J. **87**:181 (April) 1945.

That the rheumatic fever syndrome constitutes a public health problem is demonstrated by its incidence in the general population, by its rank as a cause of death in a most important age group and by the social and economic factors that place its control beyond the means of the average family.

Both voluntary and official agencies are increasing their capacities to cope with this problem, and the cooperation of the medical profession in aiding and implementing these programs and in stimulating public interest is as important as continued research into the causes, modes of spread and treatment of the rheumatic fever syndrome.

AUTHOR'S SUMMARY.

RHEUMATIC FEVER IN CHILDHOOD. H. WILLIAM ELGHAMMER, Illinois M. J. **87**: 187 (April) 1945.

In the child, rheumatic fever presents its most typical symptoms as well as its greatest variety of manifestations. The term "rheumatic infection" may be more nearly correct and may lead to a better understanding of the disease. The present view of the pathogenesis of rheumatic infection may be expressed as follows: Some persons conditioned by heredity and environment respond in a particular manner to hemolytic streptococcus infections and their reaction rather than a specific etiologic organism produces the clinical and pathologic picture. The pathology of rheumatic infection has undergone an interesting evolution. Not until recently has the involvement of the myocardium been fully appreciated. Many of the symptoms and signs are due to the toxemia produced by the infection and differ therefore in no way from toxic stages of other infections. The diagnosis of rheumatic infection in its systemic phase is often rather difficult. With the aid of the sedimentation rate, acute infections may be excluded. Studies of the blood will help to eliminate blood dyscrasias and other infections.

BARBOUR, Peoria, Ill.

THE TREATMENT OF THE RHEUMATIC SYNDROME. PHILLIP ROSENBLUM, Illinois M. J. **87**:189 (April) 1945.

Since there is no known successful remedy for rheumatic heart disease, the best management depends as much on the public health facilities which are available and the intelligent use of them as on the use of medication or other specific therapeutic agents. This involves an adequate program of proper educational and vocational guidance.

Rest is the most important single therapeutic measure. Drugs have but little influence on the inflammatory process and are of value only in certain passing phases. Treatment by vaccines, serums or sulfonamide compounds has proved disappointing. Penicillin is of no value against rheumatic fever. Salicylates are the most important drugs for use during the active stage of the disease. Large doses (120 to 150 grains [7.8 to 9.7 Gm.] daily) are usually necessary. One should be alert for toxic reactions to the salicylates. The diet should be adequate, high in vitamins and easily digestible. Sedatives are helpful for the relief of pain and for facilitating rest.

Resumption of activity by the patient should be gradual and under constant scrutiny. Measures that improve the general health are essential. Sulfonamide compounds may be given between September and June to prevent recurrent attacks. Before the disease can be considered inactive the following criteria are desirable: a normal rectal temperature, a normal pulse and a normal white blood cell count for ten consecutive days in the absence of antirheumatic drugs; the hemoglobin content and the erythrocyte count should improve, and the sedimentation time should have returned to normal or nearly normal.

Prevention is the keynote of pediatric endeavor. If physicians would see to it that every child who recovers from acute illness, mild or severe, is examined two or three weeks later, a good many with rheumatic manifestations would be detected earlier and preventive measures could be instituted in an endeavor to prevent more serious sequelae.

BARBOUR, Peoria, Ill.

IMMUNOLOGICAL STUDIES ON THE PSITTACOSIS-LYMPHOGRANULOMA GROUP OF VIRAL AGENTS. MAURICE R. HILLEMAN, J. Infect. Dis. **76**:96 (March-April) 1945.

By the intraperitoneal inoculation of chickens, neutralizing antiserums of relatively high titer and sharp specificity were prepared against the viruses of meningopneumonitis, lymphogranuloma venereum and mouse pneumonitis (Chicago). Either infected mouse lung or yolk sac appeared to be effective as an immunizing

antigen, but injection of infected mouse brain failed to produce a neutralizing antiserum to lymphogranuloma venereum virus.

It was possible to work out quantitative methods of determining the potency of the antiserums, and titers could be readily duplicated in repeated tests. More exact determinations were possible when the serum-virus mixtures were inoculated intranasally than when given intracerebrally, but definite neutralization occurred by either method.

A study of the relationships within the psittacosis-lymphogranuloma group by means of cross serum neutralization tests was made. Meningopneumonitis, psittacosis, ornithosis, human pneumonitis strains (S-F), feline pneumonitis (Baker), lymphogranuloma venereum and the Chicago and Atherton II strains of mouse pneumonitis were included. Meningopneumonitis and ornithosis viruses were found to be similar to each other and different from the other agents of the group tested. The Chicago and Atherton II strains of mouse pneumonitis also proved to be similar to each other and different from the other agents tested. None of the other agents tested showed any relationship to the three antiserums used. The relations established in the present studies by the serum-neutralization test are in agreement with those established on other bases by other workers. These results indicate that neutralization tests with immune chicken serums offer a satisfactory method for studying the relations within and identifying the members of the psittacosis-lymphogranuloma group.

TOOMEY, Cleveland.

A FIELD STUDY OF LATENT TULAREMIA IN RODENTS WITH A LIST OF ALL KNOWN NATURALLY INFECTED VERTEBRATES. A. L. BURROUGHS, R. HOLDENFRIED, D. S. LONGANECKER and K. F. MEYER, J. Infect. Dis. **76**:115 (March-April) 1945.

1. Latent tularemia infection was found in Rattus norvegicus, Microtus californicus and Peromyscus maniculatus. None of their ectoparasites were infected.

2. The possibility of an aerogenic transmission of tularemia among some rodents is suggested.

3. A list of vertebrates known to be naturally infected with tularemia is included.

AUTHORS' SUMMARY.

THE EFFECT OF CHLORINATION ON THE VIRUS OF EASTERN EQUINE ENCEPHALOMYELITIS IN THE PRESENCE OF ORGANIC SUBSTANCES. J. EMERSON KEMPF, MARJORIE E. PIERCE, MARTHA G. WILSON and MALCOLM H. SOULE, J. Infect. Dis. **76**:120 (March-April) 1945.

Suspensions of mouse brain tissue infected with Eastern equine encephalomyelitis (1 to 1,000 M. L. D.'s) in distilled water, prepared so that there was no chlorine demand, were consistently inactivated by 0.75 to 0.90 parts per million of chlorine (orthotolidine indicator); 0.68 to 1.10 parts per million (starch-iodide indicator). The potency of the virus seemingly was not involved between values of 1 and 1,000 M. L. D.; more virulent material was not studied. When the distilled water suspensions were mixed with 0.75 per cent of monkey spinal cord, 0.01 and 0.02 per cent gelatin or river water, an increase in chlorine concentrations was essential for inactivation. Since these substances all had definite chlorine demands, the virus was suspended in river water, and the break-point technic of chlorination as carried out in municipal practice was studied. The findings were inconsistent, but it could be concluded that water containing chlorine in excess of the amount required to produce a break-point was destructive to the virus.

Suspensions of the virus in distilled water were inactivated with chlorine, and then an attempt was made to reactivate the virus by the addition of sodium thiosulfate or by dilution. The results were negative.

AUTHORS' SUMMARY.

ISOLATION OF AN AGENT BELONGING TO THE PSITTACOSIS-LYMPHOGRANULOMA GROUP OF VIRUSES. ALICE H. KEMPF, ALBERT H. WHEELER and W. J. NUNGESTER, J. Infect. Dis. **76**:135 (March-April) 1945.

The isolation of a virus and some of its properties have been described. This virus appears to be another member of the psittacosis-lymphogranuloma group.

The already well recognized difficulties of determining the relationship among the viruses of this group and the possible identity of some of the known members have been reemphasized.

AUTHORS' SUMMARY.

CAUSALGIA AND GANGRENE: RARE COMPLICATIONS IN MENINGOCOCCAL MENINGITIS. PHINEAS BERNSTEIN, New England J. Med. **230**:482 (April 20) 1944.

In the enormous recent literature dealing with meningococcic meningitis, no reference to gangrene of the skin and toes terminating in spontaneous amputation or to the more unusual syndrome of causalgia has been observed. In the case reported, that of a 34 year old woman, these conditions became manifest after the initial fulminating attack subsided and caused protraction of disability for four months. That there was a common etiologic basis for these pathologic changes is uncertain and difficult to maintain.

The emboli that probably caused these phenomena were of profound clinical significance because of the possibility of their fortuitous destination in vital areas of the body—brain, heart and lungs. The prognosis was therefore guarded.

GENGENBACH, Denver, Colo.

SULPHONAMIDE FOR SONNE DYSENTERY. R. SWYER and R. K. W. YANG, Brit. M. J. **1**:149 (Feb. 3) 1945.

A series of 179 patients suffering from bacteriologically proved Sonne dysentery have been treated with sulfanilylbenzamide, sulfanilylamidobenzamide or succinylsulfathiazole in doses governed by the patients' weights. The stools became formed, normal in color and free from blood or mucus in twenty-four to forty-eight hours in the series in which sulfanilylbenzamide was used, and in 38 of these cases bacteriologic clearance was obtained in an average of 1.8 days. Improvement in characteristics of stools was decided with sulfanilylamidobenzamide but rather less so than with sulfanilylbenzamide. The average time for clearance was 3.6 and 2.5 days, with full and half doses respectively. With succinylsulfathiazole, there was not a great improvement in stools, but clearance time was 2.4 days. Bacteriologic relapse rates were with sulfanilylbenzamide, 7.3 per cent; sulfanilylamidobenzamide, full dose 15 per cent and half dose 3.3 per cent, succinylsulfathiazole, 34.6 per cent.

BIRDSONG, University, Va.

Chronic Infectious Diseases

EFFECT OF THE ORAL ADMINISTRATION OF THYMOL ON EXPERIMENTALLY INDUCED TUBERCULOSIS. RALPH MCBURNEY, LOUISE CASON and H. B. SEARCY, J. Lab. & Clin. Med. **30**:32 (Jan.) 1945.

Thymol was shown to exert a strong inhibitory action on virulent human strains of Mycobacterium tuberculosis in guinea pigs. Each guinea pig was inoculated with 0.0005 mg. of organism, and the daily dose of thymol was 15 grains (0.97 Gm.) per hundred pounds (45.4 Kg.) of body weight. Fourteen weeks following inoculation, when all untreated animals serving as controls had died from generalized tuberculosis, 70 per cent of the treated animals were living and apparently well. The authors suggest that thymol as a therapeutic agent against experimentally produced tuberculosis in animals and early recognized human pulmonary tuberculosis is worthy of further investigation and clinical trial.

WILKINSON, Galveston, Texas.

OCULAR TUBERCULOUS ALLERGY. B. V. RE, Arch. am. de med. **20**:1 (March) 1944.

The author gives a discussion of the most widely accepted theories which have been advanced to explain the pathogenesis of allergic phlyctenulosis. Of 150 patients with this condition observed at the Fiorito Hospital in all reaction to tuberculin was noted. A similar experience has been obtained by other authors.

These facts and other theoretic reasons support the thesis that this is usually an allergic response of a sensitized conjunctiva to tuberculoprotein.

ABALLI, Habana, Cuba.

Diseases of Blood, Heart and Blood Vessels and Spleen

HEART DISEASE IN THE SOUTH: I. A STATISTICAL STUDY OF 1,045 CARDIAC DEATHS. A. B. HOLOUBEK, Am. Heart J. **29**:168 (Feb.) 1945.

The author reports observations at autopsy in 1,045 cases of heart disease at the Charity Hospital of Louisiana in New Orleans over a period of six years. She found that 11.1 per cent of the deaths were due to rheumatic heart disease. This incidence of rheumatic heart disease was found to be higher than that given in some other reports from the Southern States but was much less than is encountered in the North.

GIBSON, Chicago.

INCIDENCE OF HEART DISEASE AND RHEUMATIC FEVER IN SCHOOL CHILDREN IN THREE CLIMATICALLY DIFFERENT CALIFORNIA COMMUNITIES. J. J. SAMPSON, P. T. HAHMAN, W. L. HALVERSON and M. C. SHEARER, Am. Heart J. **29**: 178 (Feb.) 1945.

The authors studied the school populations of three California communities with entirely different climates for the incidence of various types of heart disease, with special reference to rheumatic heart disease. It was observed that in the warm, dry climate of the Redlands rheumatic fever and rheumatic heart disease occurred in a degree comparable to that in states with mild temperate climates. In Susanville, a mountain community with average humidity and precipitation but with wide extremes of average winter and summer temperatures, the incidence was very high, comparable to that in northeastern United States or in Great Britain. In Eureka, with a uniformly cool climate and high precipitation, there was an unusually high incidence of rheumatic fever.

GIBSON, Chicago.

THE INCIDENCE OF HEART DISEASE IN PUERTO RICO: STATISTICAL ANALYSIS OF 1,081 CASES. R. M. SUÁREZ, Am. Heart J. **29**:339 (March) 1945.

The author has presented statistical studies based on 1,081 cases of heart disease. He found that in this group the incidence of rheumatic heart disease was 17.4 per cent. It was his opinion, based on this study, that climate per se is not an outstanding factor in the prevalence of rheumatic heart disease.

GIBSON, Chicago.

THE TREATMENT OF SUBACUTE BACTERIAL ENDOCARDITIS WITH PENICILLIN. JAMES E. PAULLIN and CRISTOPHER JOHN MCLAUGHLIN, Ann. Int. Med. **22**:475 (April) 1945.

Six cases of subacute bacterial endocarditis are reported, in 3 of which the patients recovered after treatment with penicillin. All patients require at least 4,000,000 units of the drug.

READING, Galveston, Texas.

PRESENT STATUS OF HEMORRHAGIC DISEASE. FREDEDICK W. MADISON, Wisconsin M. J. **43**:688 (July) 1944.

Determination of the site of the defect in hemorrhagic disease can be made in the great majority of cases by the use of six simple tests: 1. The Lee and White method of determining coagulation time. This time is abnormal in persons with hemophilia and hypoprothrombinemia and sometimes with thrombopenic purpura. 2. Determination of prothrombin time. This is prolonged in persons with hypoprothrombinemia. 3. Determination of clot retraction time and (4) platelet count. Clot retraction time and platelet count are roughly parallel and are abnormal in persons with thrombopenic purpura. 5. Determination of bleeding time. This is roughly a measure of capillary contractility and is prolonged in persons with thrombopenic purpura, hereditary hemorrhagic telangiectasia and pseudohemophilia. 6. Tourniquet test. This measures capillary permeability, and results are abnormal in persons with purpura, both thrombopenic and nonthrombopenic, and with scurvy.

Hemorrhage due to hypoprothrombinemia may be controlled by vitamin K or, in patients with hepatic disease, by transfusions. Purpura is rarely a serious clinical problem unless associated with a defect of coagulation or of clot retraction in addition to the capillary permeability. It is often of allergic origin. Transfusions, splenic irradiation and even splenectomy may be required if the hemorrhage is severe.

BAIRD, Wauwatosa, Wis.

MACROCYTIC ANAEMIA IN CHILDREN. L. J. DAVIS, Arch. Dis. Childhood **19**:147 (Dec.) 1944.

Three cases of megaloblastic anemia in children aged 12, 13 and 15 years are reported, and the pathogenesis in these cases is considered.

Although the blood and sternal marrow pictures were typical of pernicious anemia, arguments are advanced against the acceptance of this diagnosis in the present cases, in all of which, it is believed, defective assimilation from the alimentary tract may have been an etiologic factor of paramount importance.

Two of the patients proved refractory to parenteral injections of liver extracts of known potency but responded promptly to proteolyzed liver administered orally.

In 1 case the macrocytic, hyperchromic anemia was preceded a year earlier by a severe hypochromic anemia.

KELLY, Milwaukee.

THE RH FACTOR AND BLOOD GROUP FREQUENCIES IN JAPANESE. J. J. GRAYDON and R. E. SIMMONS, M. J. Australia **1**:601 (June 16) 1945.

The blood of 399 of 400 Japanese tested was Rh positive; only one sample was found to be Rh negative with all specimens of human anti-Rh test serum used.

GONCE, Madison, Wis.

ADELAIDE LOCAL BOARD OF HEALTH SURVEY OF SCHOOL CHILDREN BY MANTOUX AND PATCH TESTS. H. K. FRY, M. J. Australia **1**:604 (June 16) 1945.

Among 1,409 children tested, 103 (7.3 per cent) reacted to the Mantoux test with a dilution of 1:1,000 of old tuberculin and 18 (1.3 per cent) with a dilution of 1:100—a total of 122 (8.6 per cent). The percentage of positive reactors was higher in the higher age groups. Sex differences were variable. Of 50 boys of all ages and 16 girls with ages up to 10 years having positive Mantoux reactions to a 1:1,000 dilution of old tuberculin, 62 (94 per cent) also reacted to the patch test. Of 37 girls aged over 10 years who reacted to the Mantoux test with a dilution of 1:1,000, 24 (65 per cent) reacted to the patch test. Only 1 child of the 18 who reacted to the Mantoux test with a dilution of 1:100 reacted to the patch test. Two patches were applied to 62 children who reacted to the Mantoux test with a dilution

of 1:1,000. Both patches produced reactions in 41 instances and failed to produce reactions in 10 instances, and one patch test produced a reaction and one failed to produce a reaction in 11 instances. Among one hundred and twenty-eight patch tests giving positive results, one of the twin disks produced a negative result in 16 cases. Readings of the results of the Mantoux test on the fifth day were found to be more efficient than readings on the second day after the injection.

GONCE, Madison, Wis.

CONSIDERATION OF A CASE OF CHRONIC CARDIAC VALVULAR DISEASE IN A CHILD. L. VELASCO BLANCO and E. SIVORI, Arch. am. de ped. **18**:51 (July) 1942.

A case of chronic cardiac valvular disease in a 14 year old boy is presented. The patient had dyspnea on exertion, deficient development, cardiac hypertrophy, a systolic thrill at the apex and a late diastolic rumble, with a presystolic murmur ending in an accentuation during the early part of systole at the apex and transmitted to the axilla. There was no cyanosis.

Roentgenologic studies confirmed the hypertrophy and showed a prominence of the pulmonary arch. The electrocardiogram revealed a right axis deviation.

A phonocardiogram demonstrated the existence of presystolic and systolic murmurs. The sedimentation rate was within normal limits. As no definite rheumatic history was elicited, the authors point out the necessity of considering several diagnostic possibilities, among which congenital anomalies are easily ruled out but pure mitral stenosis (Durosier's syndrome) and pure mitral insufficiency deserve a careful consideration. The final diagnosis of mitral insufficiency and stenosis is strongly supported by the phonocardiogram. ABALLI, Habana, Cuba.

PATENT DUCTUS ARTERIOSUS (DUCTUS BOTALLI): ITS SURGICAL TREATMENT. O. IVANISSEVICH, L. VELASCO BLANCO and E. SAUBIDET, Arch. am. de med. **20**:15 (June) 1944.

The case of a 6 year old child with a patent ductus arteriosus, which was successfully ligated, is described. The patient showed a typical machinery murmur and a thrill, with maximum intensity at the second left intercostal space; there were a prominent pulmonary conus, a moderate hypertrophy of the left ventricle and an increased pulse pressure. The thrill disappeared immediately after the ligature, and the diastolic pressure went up 20 mm. of mercury. Nothing is specified about the murmur.

ABALLI, Habana, Cuba.

Diseases of the Gastrointestinal Tract, Liver and Peritoneum

THE PREVENTION AND ATTENUATION OF INFECTIOUS HEPATITIS BY GAMMA GLOBULIN: PRELIMINARY NOTE. JOSEPH STOKES JR. and JOHN R. NEEFE, J. A. M. A. **127**:144 (Jan. 20) 1945.

During an epidemic of infectious hepatitis in a summer camp, gamma globulin was given intramuscularly in doses of 0.15 cc. per pound (0.5 Kg.) to 53 of 331 persons between the ages of 5 and 40 years who showed no signs of disease at the time of the injection. Results indicate that the material has a significant effect in preventing or attenuating the infectious hepatitis. Best results were obtained when the globulin was injected early in the incubation period, but it seems possible that there may be therapeutic value from injections early in the preicteric stage of the hepatitis.

BAIRD, Wauwatosa, Wis.

PREVENTION OF INFECTIOUS HEPATITIS WITH GAMM GLOBULIN. W. P. HAVENS JR. and JOHN R. PAUL, J. A. M. A. **129**:270 (Sept. 22) 1945.

During an epidemic of infectious hepatitis in a children's home, intramuscular injection of gamma globulin into 97 children in doses averaging 0.08 cc. per pound

(454 Gm.) resulted in the appearance of only 2 cases of jaundice, both within six days of injection, and 6 cases of hepatitis without jaundice. Among 155 children not inoculated, there were 36 cases of jaundice and 14 cases of hepatitis without jaundice.

BAIRD, Wauwatosa, Wis.

AN ANALYSIS OF THE DUODENAL DRAINAGE IN THE STEATORRHEAS. HERBERT F. PHILIPSBORN JR., GRACE LAWRENCE, STANLEY GIBSON and HARRY GREENGARD, J. Pediat. **26**:107 (Feb.) 1945.

The clinical syndromes of celiac disease and fibrocystic disease of the pancreas may closely resemble each other. The usual laboratory procedures (twenty-four hour fecal analysis and vitamin A absorption curve) are frequently of no differential value. Determination of the cause of any one of the steatorrheic states may depend on an analysis of the enzymatic activity of the duodenal drainage.

Detailed methods by which the duodenal contents may be obtained and quantitatively analyzed for enzymatic activity are presented.

Normal values for the activity of trypsin, amylase and lipase in children are stated.

The patients studied fell into four groups: (*a*) normal children (afebrile, in a good nutritional state and without evidence of gastrointestinal disturbances), (*b*) children called "feeding problems" (those failing to gain weight and to maintain an adequate state of nutrition in spite of the fact that they were free from any apparent pathologic processes), (*c*) children with fibrocystic disease (proved either by microscopic examination or by the presence of characteristic clinical findings and a response to substitution therapy), (*d*) children with celiac disease. (These presented either all or a large majority of the criteria by which Gee first described the disease.)

The values for enzymatic activity in the duodenal juice of the normal children studied were within the expected range. The volumetric output of the pancreas was decidedly increased after stimulation by intravenous administration of secretin.

Children described as "feeding problems" had significant reduction or absence of enzymatic activity in the material obtained. That obtained from 1 of these children nine months after the original study revealed abundant enzymatic activity.

Children with fibrocystic disease of the pancreas showed permanent hypochylia or achylia pancreatica. The pancreas of these children did not respond to stimulation by secretin. This is interpreted as an intrapancreatic block which prevents pancreatic fluid and enzymes from reaching the bowel. A case of pancreatic insufficiency with infantilism is contrasted to show that in this condition the pancreatic enzymes are diminished but the volumetric response to a pancreatic stimulus is not obliterated.

Children with celiac disease have normal enzymatic activity in their duodenal juice, and the pancreatic response to secretin cannot be differentiated from that noted in normal children.

The authors believe that the analysis of the duodenal drainage in selected cases of steatorrhea will yield knowledge that will be of value in diagnosis, management and prognosis.

AUTHORS' ABSTRACT.

PARENTERAL REPAIR OF DEHYDRATION. S. Z. LEVINE, J. Pediat. **26**:196 (Feb.) 1945.

By definition, dehydration signifies a reduction in total volume of body fluids, often with alterations in their chemical composition. The two chief aims of parenteral therapy are therefore replenishment of depleted reserves by the provision of fluid over and above maintenance needs and correction of electrolyte and colloid distortions when present. The magnitude of the negative water bal-

ance determines the amounts of fluid needed in treatment, the concurrent loss of solutes, both qualitative and quantitative, and the types of solutions.

Clinical signs of dehydration in infants become manifest when fluid depletion approximates from 8 to 12 per cent of the body weight (80 to 120 cc. per kilogram). More extreme states result in greater losses (200 cc. per kilogram or more daily in severe diarrhea). In general 200 to 250 cc. of total fluids per kilogram of body weight, rarely more, by the parenteral route will suffice during the initial one or two days of starvation and 140 to 150 cc. per kilogram per day, thereafter. Hypodermoclyses and intravenous infusions are preferred over the intraperitoneal route; treatments may be intermittent at four to six hour intervals or by continuous intravenous drip.

The types of fluids selected for treatment depend on the causation of the dehydration, the relative amounts of water and solute losses and the condition of the patient. Correction of electrolyte distortion leading to metabolic acidosis may be achieved directly or indirectly. The direct approach, with use of a sixth-molar lactate solution, is favored when acidosis is due primarily to loss of fixed base (low plasma bicarbonate and low chloride levels). The indirect approach, by treatment with intravenous injection of hypertonic dextrose solution preceded by saline clysis, is preferred when the acidosis is associated with a lowered plasma bicarbonate and an elevated chloride. For severe acidotic states the combined approach is optimal. Following correction of the acidosis, body dehydration per se is treated by subsequent clyses and infusions of isotonic solution of dextrose and sodium chloride, with transfusions of whole blood or plasma to correct complicating anemia or hypoproteinemia respectively. The importance of reinstituting oral feedings as early as is feasible is stressed.

AUTHOR'S ABSTRACT.

RECURRENT APPENDICEAL ABSCESS. THOMAS F. ROSE, M. J. Australia **1**:659 (June 30) 1945.

The author reports 13 cases of recurrent appendical abscess. The average age was 30 years, the youngest patient being 7 years and the oldest 60 years. In each instance this was the first recurrence, and the intervals between abscesses averaged one hundred weeks, the shortest period being four weeks and the longest ten years. At the time of the original illness, 4 patients underwent drainage of the abscess, with complete (2) or incomplete (2) removal of the appendix; 6 underwent drainage of the abscess only, and 3 had no operation. In each instance, it was evident from the history and later observations that the cause of the original abscess was rupture of the appendix, the seat of acute obstructive appendicitis. Obstruction was caused in 9 instances by impacted fecaliths, in 1 by a carcinoid tumor and in 1 by a fishbone. The cause of the obstructions was not apparent in 2 cases. The commonest cause of recurrent abscess was further inflammation in a still obstructed, perforated appendix; this was observed in 9 cases. Free lying fecaliths were the cause in 2 cases, though the appendix had been previously completely removed. Incomplete removal of the appendix was the cause in 2 cases. In every case, the abscess recurred in its original position, being retrocecal in 11 cases and pelvic in 2. Of the 13 patients, 2 who were unable to receive operative treatment died of suppurative pylephlebitis.

GONCE, Madison, Wis.

INTUSSUSCEPTION DUE TO MECKEL'S DIVERTICULUM. L. VELASCO BLANCO and E. M. ECHEGARAY, Arch. am. de med. **18**:45 (July) 1942.

A case of intussusception due to Meckel's diverticulum in a 2½ year old boy is reported. In spite of the fact that the patient was operated on forty-eight hours after the onset, reduction of the intussusception was easily performed. Resection was limited to Meckel's diverticulum. The patient died five hours after the operation.

ABALLI, Habana, Cuba.

COMMENT ON A NEW CASE OF HYPERTROPHIC PYLORIC STENOSIS TREATED SUCCESSFULLY BY PYLORODUODENAL GAVAGE. L. VELASCO BLANCO, Arch. am. de med. **20**:45, 1944.

The author treated a 1 month old infant with characteristic symptoms of hypertrophic pyloric stenosis by medical means, which included antispasmodics, phenobarbital, thiamine, fluids by retention enemas, and feedings of breast milk by a catheter introduced as far as the pyloric antrum (Hess's technic). After a poor start the baby improved slowly and finally recovered.

This is the ninth patient for whom he has employed this form of therapy, and a cure has been obtained in every instance (100 per cent). The condition is rare in Argentina, as only these 9 children with it have been observed out of 50,000 hospitalized children registered in the author's hospital services.

ABALLI, Habana, Cuba.

MESENTERIC CYSTS — ENTEROCYSTOMA. P. L. MIRIZZI, A. P. CINELLI and L. FERRARIS, Rev. argent. norteam. de cienc. méd. **1**:373 (July) 1943.

The authors report a case of enterocystoma in a girl 5 years old. An operation was performed, and the recovery was complete. A classification and discussion of the conditions accompany the article.

GRULEE, Evanston, Ill.

THE USE OF TRIVALENT ARSENICAL COMPOUNDS IN THE TREATMENT OF STOMATITIS. L. DEL TORO RIOS, Rev. mex. de pediat. **13**:185 (Sept.-Dec.) 1944.

The author treated 26 patients with stomatitis by intramuscular injections of sulfarsphenamine. The dose employed varied from 0.01 to 0.06 Gm., and these were given from one to three times during the course of the illness. Of the total of 26 patients, 10 had ulcerative stomatitis, 12 had aphthous stomatitis and 4 had "impetiginous" stomatitis. In general the results are considered very satisfactory, as all the patients who were adequately followed up recovered completely within eight days. The average duration of illness was four days. The best results were seen in the last two varieties of stomatitis mentioned.

The diversity of opinion of different authorities is pointed out.

ABALLI, Habana, Cuba.

Nervous Diseases

HYPOTHALAMIC ATTACKS WITH THALAMIC LESION: I. PHYSIOLOGIC AND PSYCHOLOGIC CONSIDERATIONS. GEORGE L. ENGEL and CHARLES D. ARING, Arch. Neurol. & Psychiat. **54**:37 (July) 1945. II. ANATOMIC CONSIDERATIONS. CHARLES D. ARING and GEORGE L. ENGEL, ibid. **54**:44 (July) 1945.

This is the report of a case of cystic softening of the dorsomedial nucleus, the internal medullary lamina and the lateral nucleus of the right thalamus in an 18 year old youth who had had attacks of coryza, chills, high fever, nausea, vomiting, oliguria, fluctuating hypertension, tachycardia and muscle cramps since 5 months of age. These attacks were provoked by anxiety or infection. The patient also had been subject to attacks of holding his breath, cyanosis and unconsciousness from the third day of life to 12 years of age. Muscular and skeletal underdevelopment were observed. He had attacks of perspiration of the forehead, followed by pallor and dizziness when he ate hot foods, such as soups.

BEVERLY, Chicago.

WHAT IS THE GUILLAIN-BARRÉ SYNDROME? FREDERIC H. LEWEY, J. Pediat. **26**: 165 (Feb.) 1945.

Two representative cases of the Guillain-Barré syndrome are clinically described and the histopathologic observations discussed. One patient was

successfully operated on for adhesive arachnitis and two diseased sacral roots removed for examination. The other patient died unexpectedly in the fourth week of the disease of an acute degeneration of the phrenic nerves.

The sacral roots in both cases showed an identical pathologic picture, namely a peracute radiculopathy of almost necrotic nature. The myelin was pulverized and in many instances completely dissolved, the axis-cylinders fragmented. No infiltration, no compound granular bodies and only a scant scar formation from the endoneurium was found. Necropsy revealed, in addition, a polyneuropathy with acute exacerbation in the phrenic nerves, cerebral arachnitis, cortical derangement, necroses in the anterior horns and degeneration in the white matter of the spinal cord, all of which had failed to produce clinical signs.

The clinical picture and the pathologic changes did not suggest that either case deserved classification under a special heading.

Guillain, Barré and Strohl had originally believed that patients with a severe but eventually benign polyneuropathy, with preferential involvement of the motor nerves, and albuminocytologic dissociation in the spinal fluid might be united in one group. In 1937 Guillain himself revoked this opinion without reservation.

It is suggested one retain, nevertheless, the designation "Guillain-Barré syndrome," which has become popular as a general heading for the various forms of polyneuropathy and radiculopathy of unkown cause, with or without encroachment on the central nervous system and with a partial block as indicated by the albuminocytologic dissociation in the spinal fluid.

AUTHOR'S ABSTRACT.

WILSON'S DISEASE. A. J. BLAZEBROOK, Edinburgh M. J. **52**:83 (Feb.) 1945.

The author reports 2 cases of a hereditary disease with symptoms resembling those of the rare clinical entity Wilson's disease. In 1 instance a boy of 17 years is known to have had encephalitis at the age of 10, following which the course was slowly progressive, with pain in the legs, slowness of speech and stiffness of the body. Throughout the years there was dysarthria and a tendency for saliva to escape from the mouth. At the last there were coarse tremors of the arms and legs and confinement to bed. Torsion spasms, which were initiated by emotional upsets, began at 15 years of age.

The second case was also that of a boy of 17 years in whom the disease developed through a period of three years after an attack of jaundice and hepatomegaly. The clinical picture was typical of Wilson's disease, and the liver was found to contain an excess of copper; there was likewise an excess found at autopsy in the basal ganglions and to a less extent in the cortex of the brain.

The first child came from a family in which a condition of somewhat similar nature had been present in various members, at least on the male side. The author states that the disease is common in domestic animals but is rare in human beings and that the congenital defect comes by way of the liver. Cunningham is cited as an authority for the statement that in human beings the average intake of copper is much beyond the requirement; the author believes that copper acts on the tissues as a powerful inhibitor of enzymic activity. There is a rich blood supply in the basal ganglions. Inability to excrete copper probably results, as far as the hepatic dysfunction is concerned, from the production of a lenticular degeneration.

NEFF, Kansas City, Mo.

A CASE OF IDIOPATHIC ACUTE DISSEMINATED ENCEPHALOMYELITIS ("ACUTE PERIVASCULAR MYELINOCLASIS"). CHARLES SWAN, M. J. Australia **2**:15 (July 7) 1945.

Without any obvious exciting factor, a boy aged 7 years, manifested signs and symptoms of involvement of the nervous system, characterized by drowsiness, changes in habits and personality, root pains and spasms and rigidity of the limbs.

Death occurred, in association with hypostatic pneumonia, about five weeks after the onset of the initial symptoms. Histopathologic examination disclosed acute disseminated encephalomyelitis, with perivascular (extra-adventitial) demyelination and its concomitant changes. The pathologic process was most evident in the white matter of the centrum semiovale and of moderate intensity elsewhere in the cerebrum and in the brain stem and cerebellum.

GONCE, Madison, Wis.

CONGENITAL CYST IN THE LEFT TEMPORAL LOBE. S. OBRADOR, F. PASCUAL DEL RONCAL and M. FALCÓN G., Arch. de neurol. y psiquiat. de Mexico **6**:385 (Sept.-Oct.) 1943.

A 27 year old woman complained of persistent frontal and occipital headache and recurrent attacks of vertigo, of five months' duration. There were a few attacks of transitory blurring of vision. A lumbar puncture made soon after the onset relieved her symptoms for one month. Occasionally she felt as though her limbs did not exist. Loss of consciousness, without convulsive movements, occurred once.

Examination showed bilateral papilledema, right homonymous hemianopsia, weakness of the lower right side of the face, a tendency to veer to the left in the Romberg position and some inward drift of the outstretched right upper limb. The patient showed mild euphoria, some intellectual enfeeblement and amnestic aphasia. There were abnormally slow waves (4 to 5 per second) of low voltage in the left temporo-occipital region and a few delta waves in the right frontal area. Pneumoencephalographic examination showed that the right ventricle was enlarged and displaced to the right and that a small amount of air was present in the posterior portion of the left ventricle, which seemed to be pushed upward and backward.

Operation showed a voluminous cyst with white, smooth walls and poor vascularization, in the subcortex of the left temporal lobe. The cyst was more than 6 cm. in diameter and was filled with cerebrospinal fluid. No tumor nodules were found. Biopsy of a small piece revealed the wall of a cystic cavity lined with ependyma; there were a few amyloid bodies and mild neuroglial proliferation in the wall. The authors consider that this cyst was congenital and that at one time it was connected with the wall of the lateral ventricle.

SAVITSKY, New York. [ARCH. NEUROL. & PSYCHIAT.]

BULIMIA ASSOCIATED WITH EPILEPSY IN CHILDREN. B. VIJNOVSKY, Rev. argent. de neurol. y psiquiat. **9**:344 (Sept.) 1944.

Vijnovsky reports 18 cases of bulimia among 66 epileptic children. The symptom had been present since birth in 10 cases, appeared simultaneously with the attacks in 4 cases, five years before the onset of attacks in 1 case and nine years after the onset of attacks in another case. In 1 case the bulimia was accompanied with polydipsia; in another an episode of intense hunger was the prodrome of an epileptic attack. In 4 cases the bulimia disappeared simultaneously with the disappearance of the attack. No increase in weight was observed in spite of the excessive intake of food. In 9 cases blood sugar levels were determined, and they were all normal (80 to 110 mg. per hundred cubic centimeters). In 2 cases pica was present. The author believes that the increase in appetite is due to an epileptogenous focus in the frontal lobe which, at the same time, inhibits lower, probably hypothalamic, centers controlling appetite.

SAVITSKY, New York. [ARCH. NEUROL. & PSYCHIAT.]

CLINICAL OBSERVATIONS ON PATHOGENICITY OF BACTERIA FROM THE PASTEURELLA-GROUP. KRUNOSLAVA TOMIĆ-KAROVIĆ and MATO IVANOVIĆ, Ann. pædiat. **163**: 177 (Oct.) 1944.

A rare case of purulent meningitis, caused by bacteria of the Pasteurella group, in a 12 month old girl is reported by the authors. Diagnosis was made bacteri-

ologically, serologically and by animal experiment (mice) intra vitam. The patient's serum had its own strain; also the laboratory strains of Pasteurella aviseptica agglutinated in titer 1:800. The clinical diagnosis was confirmed by postmortem examination. Clinically, the case showed an acute commencement, the usual general symptoms of a purulent meningitis and a lethal course after twenty-six days. On the nineteenth day of the illness, diminution of the liver under icterus developed, which at the postmortem examination was defined as degeneratio parenchymatosa hepatis gravis. Treatment (lumbar puncture, blood transfusion and administration of sulfacetimide, methenamine, and chloral hydrate) was without success. Etiologically, an inferiority of the central nervous system (syphilis in both parents) and a possible infection by a hen must be considered.

AUTHORS' ABSTRACT.

Psychology and Psychiatry

AN INTERBEHAVIORAL ANALYSIS OF SEVERAL ASPECTS OF STUTTERING. HARRIS HILL, J. Gen. Psychol. **32**:289, 1945.

In this discussion stuttering behavior has been placed in an interbehavioral framework, the organism, with its historical backgrounds, interacting with objects, conditions or activities in a particular field, constituting the subject matter of such a study. The author believes that stuttering blocks are occasioned originally by direct stimulation, either difficulty in pronunciation and word finding or interruptions induced by affective and emotional reactions. Such blocks become substitute responses as conditioning develops. In order to understand the learning of stuttering, the author inspects the attention, perception and consummatory responses of the stutterer, his feeling and emotional behavior segments and his relationship to the person spoken to and the object spoken about.

POLLACK, Evanston, Ill.

Diseases of the Genitourinary Tract

ABDOMINAL PAIN DUE TO UROLOGIC DISEASE IN CHILDREN. MEREDITH F. CAMPBELL, J. A. M. A. **128**:326 (June 2) 1945.

Acute abdominal pain in children usually requires surgical consideration and often laparotomy. The child with persistent or recurrent abdominal pain should have a complete examination, including urologic investigation, before being subjected to laparotomy.

Pain of urologic origin in children is usually due to obstruction, with distention of the collecting part of the urinary tract. Superimposed infection intensifies the clinical manifestations. Symptoms may be predominantly gastrointestinal. A normal urine does not rule out urologic disease.

Congenital anomalies of the urogenital tract are common, especially ureteral obstructive lesions. If on the right side, these may be erroneously diagnosed as "chronic appendicitis." Excretory urograms will often establish the diagnosis, but if any doubt remains cystoscopy, ureteral catheterization and retrograde pyelography are necessary.

BAIRD, Wauwatosa, Wis.

SULFAMERAZINE IN THE TREATMENT OF GENITO-URINARY INFECTIONS: AN ANALYSIS OF 151 CONSECUTIVE CASES. W. E. KITTREDGE and ARTHUR J. BUTT, New Orleans M. & S. J. **98**:63 (Aug.) 1945.

Reporting on the use of sulfamerazine in 151 cases of urinary infections, the authors conclude that: (1) the drug is efficient in relatively small doses; (2) its influence is exerted in a relatively short time; (3) it shows no higher incidence of reactions than comparable doses of other sulfonamide compounds; (4) it may be tolerated when other forms are not; (5) it may be effective when other forms fail, and (6) it is not effective against gonorrhea.

BERKLEY, Beverly Hills, Calif.

Diseases of the Ductless Glands; Endocrinology

THE SYNDROME OF PRECOCIOUS PUBERTY, FIBROCYSTIC BONE DISEASE AND PIGMENTATION OF THE SKIN: ELEVEN YEARS' OBSERVATION OF A CASE. BERNARD M. SCHOLDER, Ann. Int. Med. **22**:105 (Jan.) 1945.

Scholder reports the case of a 19 year old woman who has been under his observation for eleven years. Delivery was attended by considerable trauma to the mother. Vaginal bleeding began suddenly in the child at the age of 3 years and recurred at irregular intervals for the next two years; since the age of 7 it has been a constant feature. Tenderness of the breast appeared at 3, and the mammae became prominent at 4 years. At the age of 5, pubic hair appeared and facial asymmetry became apparent. The syndrome of precocious puberty, fibrocystic changes in bone and pigmentation of the skin has been compatible with an otherwise normal life and development. Physical examination revealed slight facial asymmetry, decrease in size of the left limbs and small stature. Growth ceased at the age of 12, with a maximum of 61.5 inches (156 cm.). The results of laboratory studies were not remarkable except for an increase in the amount of "sex hormone" in the urine when the patient was 9 years of age. Roentgenographic studies have shown profound and widespread changes in bone condensation and absorption, but there has been only slight progression of these changes over the past ten years.

Scholder advances the hypothesis that the syndrome results from a hypothalamic (pituitary) parathyroid disturbance.

GUTTMAN, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

THE TREATMENT OF ADDISON'S DISEASE BY THE IMPLANTATION METHOD. C. F. KEMPER, Ann. Int. Med. **22**:161 (Feb.) 1945.

After a stabilization period during which sodium chloride and daily injection of desoxycorticosterone are given, implantations of pellets of desoxycorticosterone acetate are used, the amount of which is based on the calculated needs of the patient. Reimplantations are made about once a year.

READING, Galveston, Texas.

Diseases of the Muscles, Bones, Joints and Lymph Glands

CALCIFICATION OF THE INTERVERTEBRAL DISCS IN CHILDHOOD. H. STEPHEN WEENS, J. Pediat. **26**:178 (Feb.) 1945.

Six cases with calcification of the intervertebral disks involving the nucleus pulposus are described. Five of these cases were recorded in the literature, and 1 was observed by us. All cases were characterized by an acute episode of pain in the region of the involved segment of the spine, limitation of motion and spinal deformity.

In the absence of pathologic studies it is not possible to arrive at definite conclusions as to the cause of this phenomenon in childhood. However, signs and symptoms of infection were noted in most of the cases and suggest that an inflammatory process was associated with the calcification. The fact that the intervertebral disks in childhood possess a blood supply may be important in this connection. The intervertebral disks are thus open to any disease carried by the blood stream. An aseptic necrotic process as a possible cause of the calcification is mentioned.

The number of observed cases is too small to create definite impressions. They suggest, however, that there exist certain differences between calcification of the intervertebral disks in childhood and in adult life.

1. Involvement of the cervical portion of the spine was noted in 4 of 6 cases occurring in childhood. Calcification of the nucleus pulposus in adult life is localized predominantly in the dorsal and the lumbar portion of the spine.

2. Rapid change in the size of the calcification or complete absorption was noted in 5 of the 6 described cases. Calcification of the nucleus pulposus in adult life is as a rule a stationary process.

3. Rather striking clinical symptoms were encountered in the cases occurring in childhood. Calcification of the nucleus pulposus in adult life is probably of little clinical significance.

In all cases complete recovery was noted.

The recognition of this syndrome appears important in view of the differential diagnosis of meningitis, myositis and diseases involving the vertebrae or spinal cord.

AUTHOR'S SUMMARY.

VASODILATATION IN THE TREATMENT OF RHEUMATOID ARTHRITIS. CHESTER M. KURTZ, O. S. ORTH and GONZALO SEPULVEDA, Wisconsin M. J. **44**:761 (Aug.) 1945.

Nicotinic acid was chosen as a therapeutic agent against rheumatoid arthritis because of its potent vasodilating properties. Thirty-six patients were treated, the youngest being 2½ years old. The drug was administered intravenously in doses of 200 to 400 cc. of 0.05 per cent isotonic solution of sodium chloride, or oral, in doses of 50 to 200 mg. every fifteen minutes for three doses, twice daily on an empty stomach. The dosage was regulated to produce a prolonged flushing of the skin. Physical therapy was given in addition to the nicotinic acid in most cases.

For 1 patient the treatment had to be discontinued because of nausea, vomiting and cyanosis. The remaining patients tolerated the drug well, some of them for as long as eleven months.

The preliminary report indicates that of the 35 patients 16 showed marked and 9 moderate subjective improvement, and 17 showed moderate and 9 marked objective improvement.

BAIRD, Wauwatosa, Wis.

TRAUMATIC DISLOCATION OF THE HIP IN A CHILD. L. VELASCO BLANCO and E. DE ECHEGARAY, Arch. am. de med. **18**:64 (Oct.-Dec.) 1942.

The rarity of traumatic dislocation of the hip during childhood is pointed out by the authors. A case of its occurrence in an 8 year old child is reported. Reduction was accomplished (with the patient under anesthesia) in spite of the fact that the dislocation had occurred ten days before. There were no complications or sequelae.

ABALLI, Habana, Cuba.

Diseases of the Eye

THE GOAL OF AN EYE-HYGIENE PROGRAM FOR SCHOOL CHILDREN. JAMES J. REGAN, New England J. Med. **231**:486 (Oct. 5) 1944.

The materials required for the visual test are a Boston Test Chart and an eye occluder which can be held by the child. The eye occluder is one devised by the Medical Department of the United States Navy. One end is blank and is held by the child over the eye not to be tested, while the other end has an opening larger than the eye, so that the teacher may observe squinting or other signs of eyestrain.

The test chart contains a series of at least four E's, the limbs of which are of equal length and width, with the open ends pointing to the right, to the left, upward and downward. There are four rows, the largest size above and the smallest below. The top row is marked 50 feet (15.2 meters) and the other rows 40 feet (12.2 meters), 30 feet (9.1 meters) and 20 feet (6.1 meters).

The child stands 20 feet from the test chart and is asked to indicate with his hand the direction in which the open ends of the limbs point, starting with the top row. Thus, if the direction of at least two of the E's on the lowest row is read correctly, he is given a mark of 20/20. Otherwise, he is given a mark on the basis

of the lowest row in which he can succeed in successfully passing the test and receives a mark of 20/30, 20/40, or 20/50.

If he cannot read correctly at least two of the E's in the top row, he is successively moved up to 15 (4.6 meters), 10 (3 meters) and 5 feet (1.5 meters) of the chart. The distance at which he succeeds in passing the test is then marked 15/50, 10/50 or 5/50. Then the other eye is tested.

The teacher, of course, also looks for other signs of eyestrain, such as sties, crusted red lids, watery or irritated eyes and headache, especially after close work. Pupils showing signs of eyestrain are referred for a medical eye examination.

GENGENBACH, Denver, Colo.

Skin Diseases; Allergy

THE SIGNIFICANCE OF HISTAMINE IN ANAPHYLAXIS. CARL A. DRAGSTEDT, J. Allergy **16**:69 (March) 1945.

In an excellent article the author reviews the evidence for and against histamine as a causal factor in the production of anaphylaxis, or allergy. There appears to be more evidence to suggest that histamine is a causal factor than to show that it is not.

HOYER, Cincinnati.

TREATMENT OF URTICARIA WITH SYNTHETIC VITAMIN K. J. H. BLACK, J. Allergy **16**:83 (March) 1945.

The author conducted a study of a group of patients with chronic urticaria, for which the cause could not be found and for which no relief was obtained by the usual methods. Synthetic vitamin K gave relief to about 62 per cent of the patients.

HOYER, Cincinnati.

"TREATMENT OF RAGWEED POLLINOSIS WITH ANTIGEN-ANTIBODY MIXTURES." MILTON B. COHEN, and HAROLD J. FRIEDMAN, J. Allergy **16**:121 (May) 1945.

Three persons sensitive to pollen were treated with a mixture of thermostabile antibody and pollen extract. The mixture was approximately neutral or contained not more than 100 units of free antigen per cubic centimeter.

The mixture stimulated the production of thermostabile antibody. Good clinical results were obtained for all 3 persons with but three or four injections of the material.

HOYER, Cincinnati.

A STUDY OF THE FUNGUS CONTAMINANTS OF THE AIR OF SAN DIEGO AND VICINITY. GEORGE F. HARSH and SONIA E. ALLEN, J. Allergy **16**:125 (May) 1945.

The incidence of air-borne fungi over San Diego and vicinity was studied over a period of three years. The gravity slide and culture plate methods were used.

One hundred and thirty-one species comprising twenty-seven genera were isolated. The major genera are listed.

In the area studied, the number of children sensitive to molds exceeded greatly the number of adults who were sensitive. There was no great difference in the two groups with respect to pollens.

HOYER, Cincinnati.

HEREDITARY HEMORRHAGIC TELANGIECTASIA: AN ANALYSIS OF CAPILLARY HEREDOPATHIES. KARL SINGER and W. Q. WOLFSON, New England J. Med. **230**:637 (May 25) 1944.

A family presenting hereditary hemorrhagic telangiectasia, together with a positive reaction to a tourniquet test, is described. Pseudohemophilia is characterized by a prolonged bleeding time but no reaction to a tourniquet test, and hereditary

purpura simplex, by a normal bleeding time but a positive reaction to a tourniquet test. Combinations of both types exist.

Hereditary hemorrhagic telangiectasia is a localized gross abnormality of capillaries usually not accompanied with any systemic capillary dysfunction. The observed family represents, however, such a combination of localized gross deviation of capillary structure and increased capillary fragility.

GENGENBACH, Denver.

A SIMPLIFIED METHOD FOR CULTURING FUNGI FROM THE SCALP. ERNST BERNHARDT, New England J. Med. **231**:703 (Nov. 23) 1944.

The author reports a method that can be readily performed at the physician's office with little expense and permits the differentiation of the microsporon fungi from each other and from Trichophyton gypseum.

The most practical formula for the culture medium was found to be as follows: rice, 5 cc.; water, 25 cc., and aqueous (1:10,000) solution of gentian violet, 0.8 cc. The only equipment required consists of a saucepan, bottles of 100 or 125 cc. capacity, a 1 cc. pipet or syringe, test tubes, a platinum loop or needle and a microscope.

Fourteen strains of Microsporon cannis and seven of Microsporon audouini were isolated and identified by this method, with checking by cultures on Sabouraud's medium.

GENGENBACH, Denver, Colo.

IMPETIGO CONTAGIOSA CURED BY FEVER. OSCAR GREEN, U. S. Nav. M. Bull. **43**: 136 (July) 1944.

Impetigo contagiosa involving the lips healed in five days following a temperature of 104.2 F. during an attack of malaria. Natural or induced fever, the author suggests, may be of possible therapeutic value for this disease.

RODIN, South Bend, Ind. [ARCH. DERMAT. & SYPH.]

Surgery and Orthopedics

BORIC ACID OINTMENT: A STUDY OF THE POSSIBLE INTOXICATION IN THE TREATMENT OF BURNS. CARL C. PFEIFFER, LOIS F. HALLMAN and ISIDORE GERSH, J. A. M. A. **128**:266 (May 26) 1945.

This study was made because of the increasing use of boric acid ointment on burns since the Cocoanut Grove disaster, where its use under pressure dressings gave favorable results. Studies on animals indicate that "boric acid, whether applied in the form of an ointment or a saturated solution to extensive wounds, is a cumulative poison." The authors suggest the use of some other, less potentially harmful, agent.

BAIRD, Wauwatosa, Wis.

Miscellaneous

DIASONE: ITS TOXICITY AND THERAPEUTIC EFFECTIVENESS. G. W. RAIZISS; M. SEVERAC, and J. C. MOETSCH, J. Lab. & Clin. Med. **30**:317 (April) 1945.

Diasone (disodium formaldehyde sulfoxylate diaminodiphenylsulfone) has been used in the treatment of experimental and clinical tuberculosis. The authors present studies of the toxicity of this drug in lower animals. Their general conclusion is that the chronic and acute toxicity of diasone, tested orally and intravenously on mice, rats and rabbits, is noticeably less than that of most well known sulfonamide compounds, including sulfadiazine. The drug was also found to have high therapeutic effectiveness in mice infected with beta hemolytic streptococci and type II pneumococci.

WILKINSON, Galveston, Texas.

Book Reviews

Clinical Roentgenology of the Digestive Tract. By Maurice Feldman, M.D., Assistant Professor of Gastroenterology, University of Maryland; Assistant in Gastroenterology, Mercy Hospital; Consulting Roentgenologist, Sinai Hospital. Second edition. Price, $7. Pp. 769, with 551 illustrations. Baltimore: Williams & Wilkins Company, 1945.

The author is at heart a student of gastroenterology who uses the roentgenologic method as his chief tool. The book reflects this approach and provides a veritable gold mine of information. The various diseases and disorders of the digestive tract are discussed in detail with a profusion of facts, figures and statistics. The illustrations are abundant, well chosen and instructive; they show the tremendous background of knowledge and experience possessed by the author. A number of rare lesions and many helpful diagrams are included. A few of the roentgenograms, however, are rather poor and obviously old. Some, such as figures 255, 256 and 258, seem definitely to have been retouched and to have suffered thereby. Relatively small use has been made of the compression and spot film technics.

Certain conditions, such as the cascade stomach, and the "unstable or irritable duodenum," are, in the opinion of the reviewer, erroneously described as entities and their alleged symptoms enumerated. The author accepts certain other questionable concepts such as spasm of the pylorus attributed to lesions elsewhere in the digestive tract, "appendicitis, cholecystic disease, colonic dysfunction, renal conditions, allergic or toxic states, endocrine disturbances and neurosis" (page 121).

One wonders also about the evidence for some of the diagnoses made, as, for instance, in the case of prolapsing gastric mucosa (figure 146) simulating gastric carcinoma. A statement as to the gastroscopic, operative or postmortem observations would have been reassuring. Likewise, the author seems to the reviewer to speak with more confidence than is justifiable of the roentgenologic diagnosis of gastritis.

Nevertheless, in spite of these relatively minor criticisms, the book is a splendid reference text and is highly recommended. The wealth of material available is evidenced by the inclusion of chapters on the gallbladder, biliary ducts, liver, pancreas, spleen, peritoneum, mesentery and retroperitoneal tumors, abdominal vessels, deficiency diseases and leukemia. The paper is excellent, the format satisfactory and the bibliography helpful.

Textbook of Obstetrics. By Henricus J. Stander, M.D. Stander's third revision. Price, $10.00. Pp. 1287, with 973 illustrations. New York: D. Appleton-Century Company, Inc., 1945.

In 1903 appeared the first edition of Williams' "Obstetrics." It was an excellent book, both for students and for practitioners, and it received an immediate and wide recognition. At the time of Whitridge Williams' death the book had gone through six editions. During the period during which these six editions had appeared, the pupils of Williams had come to occupy university positions and important hospital positions all over the country. When a new edition was needed, one of these pupils took up the task of his former teacher. Since then, three editions have appeared from the hand of Dr. Stander, of which this is his third revision and the last.

The book is so widely known that extended comment would seem unnecessary. It is one of the two leading textbooks on obstetrics of the United States, and it is probably used in more schools than any other single book. If wide use as a textbook may be regarded as an evidence of work, and it would seem that it is a fair measure of value, the book has proved itself.

The book, together with its index, contains 1,277 pages. The last edition occupied 1,400 pages. It is a brave editor who can delete and abbreviate. The

organization of the book has been somewhat changed, the contents being presented in sections and subheadings instead of in chapters. This, the author believes, will facilitate later revisions. As the last three editions have followed one another, the experience of the Cornell Clinic and the New York Lying-In Hospital has gradually replaced that of the Johns Hopkins Hospital, on which the earlier editions were based.

The author's ability in chemical research is reflected in the chapter on the toxemias, and this section of the book is a valuable one. The discussion of the management of pregnancy and labor in the presence of cardiac disease is instructive, and the rational conservatism of the text will meet the approval of teachers of obstetrics.

The portion devoted to operative obstetrics is excellent. Discussion of the indications for the various procedures and the technic required for their performance is clear and accurate.

The chapter devoted to ectopic and abdominal pregnancy may be commended, and the discussion of the mechanism of labor in the contracted pelvis is well done.

When Dr. Williams brought out the first edition of the book, he was fortunate in that Max Broedel, the medical illustrator, was in Baltimore, and many of the illustrations were by him. Some of these remain in the present edition, and the author is commended for his judgment in continuing to employ them, for there are none better. Many new illustrations appear, some of which are by Miss E. H. Broedel. The book is liberally illustrated, and the illustrations are of a high quality. The wise and judicial tone of the book may be indicated by the caution in the chapter on puerperal infection, in which, after a sane discussion of chemotherapy, the reader is warned not to leave all to the "chemotherapeutist."

One does not often see a book which may be more fully commended. It has already had a long and useful life. This edition should prolong its usefulness, and it will no doubt be welcomed in many classrooms as well as in the libraries of many obstetricians.

Review of Essentials of Body Mechanics in Health and Disease. Fourth edition. By Joel E. Goldthwait, M.D., LL.D.; Lloyd T. Brown, M.D.; Loring T. Swaim, M.D., and John G. Kuhns, M.D., with a chapter on "The Heart and Circulation as Related to Body Mechanics," by William J. Kerr, M.D. Price, $5.00. Pp. 337, with 128 illustrations. Philadelphia: J. B. Lippincott Company, 1945.

Because of the general interest in physical fitness, the publication of this fourth edition of a book on body mechanics is of special interest. It is a standard book dealing with fundamental subjects, significant to all branches of medicine. It is well written by authorities on the subject. The illustrations and the type of paper and print are clear and good. The authors point out that the relationship of the body mechanics plays an important part in the prevention and cure of many diseases and dysfunctions of the bodily systems. The content is divided into chapters which deal with the various physical types of bodies, the body mechanics with the development of physical deformities and their association with the development of diseases of the various physiologic systems.

Although medical ideas come and go, there should be no argument concerning the influence of body mechanics on the general state of health of a person. Although, generally, too little attention is given to their influence, perhaps this publication will stimulate a wider application of fundamental principles which merit more support. In the development of the young, in the maintenance of good health in the adult and in the palliation of degenerative diseases in the old, body mechanics have a specific influence.

This book should attract the attention of students of medicine of various ages. Not only should the specialist in medicine and orthopedic surgery be interested, but general practitioners should find it extremely interesting and valuable, as the relationships between posture and physiologic disturbances are demonstrated, with outlines for proper postural treatment.

Hayfever Plants: Their Appearance, Distribution, Time of Flowering and Their Role in Hayfever, with Special Reference to North America (New Series of Plant Science, Vol. 15). By Roger P. Wodehouse, Ph.D. Price, $4.75. Pp. 245, with illustrations. Waltham, Mass.: Chronica Botanica Co.; New York: G. E. Stechert & Company, 1945.

Allergists have indeed been fortunate for many years in having the close cooperation of a botanist of Dr. Wodehouses' caliber and industry. In this book he has gathered the results of a large amount of study concerning the plants which cause hay fever. The first one hundred and sixty-three pages are devoted to a systematic discussion of the grasses, weeds and trees, with many excellent line sketches to aid in identifying the various species. The last third of the book is devoted to regional surveys. The United States is divided into ten regions, and a list of plants and their pollinating times is given for each region.

One is impressed by the great variability of flora in the different regions, and particularly by the large number of plants which may cause hay fever in the Southern and in the Southwestern States. Inasmuch as it is necessary for an allergist to be thoroughly familiar with all plants causing hay fever, Dr. Wodehouse's book will be invaluable to those practicing in any area which has a large and variegated hay fever flora.

This book is set in a superb type with which the reviewer is not familiar and is unusually well designed and arranged. Whoever designed it is a master in his art.

This is a scholarly, well written, scientific book. It is a reference book rather than one to read through, and as such it will be of considerable value to the allergist. It holds little of interest to the average physician or medical student.

How a Baby Grows. By Arnold Gesell, M.D. Price, $2. Pp. 77, with over 800 photographs arranged and interpreted with the assistance of Katherine Gesell Walden. New York: Harper & Brothers, 1945.

This book constitutes an unusual approach to the growth and development of the child. Other texts satisfy themselves with descriptions of what the child does under normal conditions; this, however, in serial pictures, shows the actual development from birth to 5 years. It is an unusual book and a useful one.

The text is taken up with descriptions of different portions of the body and how they develop and with different activities. It stresses the fact that children are not always the same and that delay in development along certain lines is oftentimes no indication of abnormality. A perfectly sane book, it can be put in the hands of mothers safely and will do much to guide them in their care of the child and to influence them along the lines of proper rearing.

Of special value is the last chapter, "A Closing Word to Parents: Enjoy Your Children."

What To Do About Vitamins. By Roger J. Williams. Price, $1.00. Pp. 56, with 34 illustrations. Norman, Okla.: University of Oklahoma Press, 1945.

This is one of the most concise, lucid and authoritative little guides on vitamins and minerals written for the layman. The author combines a wholesome amount of common sense with scientific fact in presenting this important subject to his readers. In his introduction he gives a simple but adequate fundamental discussion of the physiology of nutrition and of the qualities of the important foods in providing a well balanced diet. He lists the necessary vitamins and minerals and briefly discusses how they may be obtained from natural foods, the particular ones that need to be watched and how losses in cooking may be avoided. The discussion of the results of improper eating, food fads, food allergies and fasting is dignified and well documented with sound evidence. The outstanding practical feature of this book is the chapters on the quantitative assessment of various foods and diets. Convenient and simple graphs, charts and tables illustrate the text and make it easy for the reader to visualize the qualitative and quantitative aspects of the

foods at a glance. A special sheet has been prepared, which may be copied by the use of a tracing paper and which enables the reader to construct analytic diagrams of the food he eats or proposes to eat. This procedure is referred to as "metering" the vitamin and mineral content of food and is a unique method of testing the reader's appreciation of the text. The little book is highly recommended as an authoritative and interestingly written treatise for the layman on vitamins and minerals that is sound and practical.

News and Comment

GENERAL NEWS

Physicians Certified by the American Board of Pediatrics, Inc.—The following physicians were certified as specialists by the American Board of Pediatrics, Inc., after the examinations in New York, Dec. 7, 8 and 9, 1945:

Charles Abler, New York; George Roland Alpert, New York; Katherine H. Anderson, Winston-Salem, N. C.; Marguerite E. Bagge, Evanston, Ill.; John Donald Bailey, Rapid City, S. D.; John B. Bartram, Philadelphia; Capt. Ralph Edmond Baxter, M.C., Coral Gables, Fla.; George Bialkin, Port Arthur, Texas; Emanuel B. Brandes, Houston, Texas; Amy Breyer, Hartford, Conn.; John P. Burgess, Rock Island, Ill.; Philip S. Chasin, Forest Hills, N. Y.; Joan Neilson Daly, Forest Hills, N. Y.; William W. Davis, Oak Hill, West Va.; Charles P. DeFuccio, Jersey City, N. J.; Joseph Henry Di Leo, New York; Herman Eisenberg, Washington, D. C.; Thomas Campbell Goodwin, Baltimore; Werner Karl Gottstein, Chicago; Lester J. Greenberg, Far Rockaway, N. Y.; Paul Hogg, Newport News, Va.; Hedwig Heda Holzer, Dorchester, Mass.; Ruth M. Kraft, Detroit; Capt. Philip J. Kresky, Sioux Falls, S. D. (formerly Brooklyn); Harvey Deppen Leinbach, Jr., Reading, Pa.; Audrey Jane McDonald, Ft. Worth, Texas; Israel Miller, Brooklyn; J. Leonard Moore, Princeton, N. J. (formerly Beirut, Lebanon); Charles Anthony Murphy, Cranston, R. I. (formerly Stamford, Conn.); James Joseph Quilligan, Jr., Ann Arbor, Mich.; Helen Seibert Reardon, Ann Arbor, Mich.; Capt. Edward T. Reilly, M.C., Minneapolis (formerly Brooklyn); Dean Winn Roberts, Baltimore; Laura Ross-Venning, Charlotte, N. C.; Robert Thornton Rutherford, Jr., Alexandria, Va.; Alec Robert Schwartz, Pittsburgh; Capt. Aaron Schwinger, M.C., Newburgh, N. Y.; Walter F. Sethney, Menominee, Mich.; Christopher Harrison Snyder, New York; Eleanor Robb Stein, Harrisburg, Pa.; Robert Hosea Trimby, Lansing, Mich.; David William Van Gelder, Baton Rouge, La.; William Lucas Venning, Charlotte, N. C.; Helen Margaret Wallace, New York; Capt. Theresa Ting Woo, M.C., Washington, D. C.; Benjamin J. Wood, Pittsburgh; Paul V. Woolley Jr., Portland, Ore.; Ella Zuschlag, Rochester, Minn.

Postgraduate Course in Pediatrics.—Michael Reese Hospital Post-Graduate School, with the cooperation of the members of the Department of Pediatrics of the University of Chicago and of Loyola University School of Medicine, offers a course in pediatrics. The course is to be held at Michael Reese Hospital from May 1 to May 29, 1946. Full time, tuition $100.

Further information may be obtained from Dr. Samuel Soskin, Dean, Michael Reese Hospital Post-Graduate School, Twenty-Ninth Street and Ellis Avenue, Chicago 16.

Directory of Pediatric Societies *

INTERNATIONAL

International Association of Preventive Pediatrics

President: Prof. S. Monrad, Dronning Louises Børnehospital, Copenhagen, Denmark.

Secretary: Dr. Daniel Oltramare, 15 Rue Lévrier, Geneva, Switzerland.

International Congress of Pediatrics

President: Dr. Henry F. Helmholz, Mayo Clinic, Rochester, Minn.

Secretary-General: Dr. L. Emmett Holt Jr., 477 First Ave., New York 16.

Canadian Committee:

Chairman: Dr. Alan Brown, Hospital for Sick Children, 67 College St., Toronto 2.

Secretary: Dr. H. P. Wright, 1414 Drummond St., Montreal.

Place: New York. Time: Postponed indefinitely.

International Congress for the Protection of Infancy

Secretary: Prof. G. B. Allaria, Corso Bramante 29, Torino 120, Italy.

FOREIGN

Argentine Pediatric Society of Buenos Aires

President: Dr. Martin Ramón Arana, 1809 Rodriguez Peña, Buenos Aires.

General Secretary: Dr. Alfredo Larguia, Cerrito 1179, Buenos Aires.

Associação Paulista de Medicina, Secçaõ de Pediatrias

President: Dr. Vicente Lara.

First Secretary: Dr. Armando de Arruda Sampaio.

Second Secretary: Dr. Luiz Augusto Monteiro Toledo, Av. Brigadeiro Luiz Antonio 393, 1° Andar, São Paulo, Brazil.

Time: Twelfth of every month, 8:30 p. m.

British Paediatric Association

President: Prof. L. G. Parsons, 58 Calthorpe Rd., Five Ways, Birmingham.

Secretary: Dr. Donald Paterson, 27 Devonshire Pl., London, W. 1.

Danish Pediatric Society

President: Dr. E. Lenstrup, Copenhagen.

Secretary: Dr. Gjørup, Dronning Louises Børnehospital, Copenhagen.

Nederlandische Vereeniging voor Kindergeneeskunde

President: Dr. J. H. G. Carstens, Servaasbolwerk 14ª, Utrecht.

Secretary: Dr. R. P. van de Kasteele, Laan van Poot 340, 's Gravenhage.

Place: Different places. Time: Three times a year.

Palestine Jewish Medical Association, Section of Physicians of Children's Diseases

President: Prof. L. F. Meyer, 29 Toelsohn St., Tel Aviv.

Secretary: Dr. A. Brünn, 9 Maazestre, Tel Aviv.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

ROYAL SOCIETY OF MEDICINE, SECTION FOR THE STUDY OF DISEASE IN CHILDREN

President: Prof. Norman Capon, 49A Rodney St., Liverpool, England.
Secretary: Dr. J. N. O'Reilly, 46 Harley St., London, W. 1, England.
Place: 1 Wimpole St., London. Time: Fourth Friday of each month, 4:15 p. m.

SOCIEDAD CUBANA DE PEDIATRIA

President: Dr. Teodosio Valledor Campo, Habana.
Secretary: Dr. Carlos Hernandez Miyares, Hospital Nuestra Señora de las Mercedes L y 21, Apartado, 2430, Habana.
Place: Cátedra de Clinica Infantil, Hospital Nuestra Señora de las Mercedes y Hospital Municipal de Infancia, Habana. Time: Last Wednesday of every month.

SOCIEDAD ECUADORIANA DE PEDIATRIA

President: Dr. Francisco de Icaza Bustamente.
Secretary: Dr. M. I. Gómez Lince, P. O. Box 693, Guayaquil.

SOCIEDAD MEXICANA DE PEDIATRIA

President: Dr. Fernando López Clares, 12/a. Medellín 191, Mexico.
Secretary: Dr. Jesus Gómez Pagola, Versalles 64, Mexico.

SOCIEDAD DE PEDIATRIA DE CÓRDOBA

President: Dr. Juan Francisco Herrera, Córdoba, Argentina.
Secretary: Dr. Bernardo Serebrinsky, Dean Funes 2000, Esq. Ocaña (N.), Córdoba, Argentina.

SOCIEDAD DE PEDIATRÍA DE SANTIAGO DE CUBA

President: Dr. Antonio Béguez César, Sagarra 107, Santiago de Cuba.
Secretary: Dra. Susana Texidó Vaillant, Padre Pico 57, Santiago de Cuba.
Place: Colegio Médico.

SOCIEDAD VENEZOLANA DE PUERICULTURA Y PEDIATRIA

President: Dr. Pedro J. Alvarez.
Secretary: Dr. Ernesto R. Figueroa, 3a Avenida 10, Bella Vista, Caracas.
Place: Hospital Municipal de Niños Jose Manuel de los Rios, Caracas.

SOCIÉTÉ DE PÉDIATRIE DE PARIS

President: Dr. Leveuf, Paris, France.
Secretary: Dr. Jean Hallé, 10 bis Rue Pré aux Clercs, Paris, France.
Place: Hôpital des Enfants Maladies, 149 Rue de Sèvres. Time: 4:30 p. m., third Thursday of every month.

URUGUAYAN SOCIETY OF PEDIATRICS

President: Dr. José Alberto Praderi, Eduardo Acevedo 1132, Montevideo.
Secretary: Dr. Héctor Fossatti, Durazno 1882, Montevideo.
Place: Club Medico del Uruguay, Avenida Agraciado 1464 (Piso 13), Montevideo.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON PEDIATRICS

Chairman: Dr. John Aikman, 184 Alexander St., Rochester, N. Y.
Secretary: Dr. Gilbert J. Levy, 188 S. Bellevue Blvd., Memphis, Tenn.
Place: San Francisco. Time: July 1-5, 1946.

AMERICAN ACADEMY OF PEDIATRICS

President: Dr. Jay I. Durand, 515 Cobb Bldg., Seattle, Wash.
Secretary: Dr. Clifford G. Grulee, 636 Church St., Evanston, Ill.
Time: Jan. 15-18, 1946.
Region I. Place: Hotel Pennsylvania, New York. Time: April 2-4, 1946.

AMERICAN HOSPITAL ASSOCIATION, CHILDREN'S HOSPITAL SECTION

Chairman: Dr. Joelle C. Hiebert, 299 Main St., Lewiston, Maine.
Secretary: Dr. W. Franklin Wood, McLean Hospital, Waverly, Mass.

AMERICAN PEDIATRIC SOCIETY

President: Dr. C. Anderson Aldrich, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Hugh McCulloch, 325 N. Euclid Ave., St. Louis 8.

CANADIAN SOCIETY FOR THE STUDY OF DISEASES OF CHILDREN

President: Dr. H. S. Little, 300 Dufferin Ave., London, Ontario.
Secretary-Treasurer: Dr. Elizabeth Chant Robertson, 67 College St., Toronto 2.
Place: Banff, Alberta. Time: June 14-15, 1946.

SOCIETY FOR PEDIATRIC RESEARCH

President: Dr. Edward M. Bridge, 219 Bryant St., Buffalo, N. Y.
Secretary: Dr. Mitchell I. Rubin, 1740 Bainbridge St., Philadelphia 46.

SECTIONAL

INTERMOUNTAIN PEDIATRIC SOCIETY

President: Dr. Robert Alway, University of Utah School of Medicine, Salt Lake City 2.
Secretary-Treasurer: Dr. L. Paul Rasmussen, 1020 Boston Bldg., Salt Lake City 1.
Place: Salt Lake City General Hospital. Time: First Thursday of each month, 8 p. m.

NEW ENGLAND PEDIATRIC SOCIETY

President: Dr. William P. Buffum, 122 Waterman St., Providence, R. I.
Secretary-Treasurer: Dr. James Marvin Baty, 1101 Beacon St., Brookline, Mass.
Place: Boston Medical Library. Time: Three meetings a year, occurring from September to May.

NORTH PACIFIC PEDIATRIC SOCIETY

President: Dr. Percy Guy, 502–2d Ave., Seattle, Wash.
Secretary: Dr. Aldis B. Johnson, 515 Cobb Bldg., Seattle, Wash.
Place: Banff, Canada. Time: June 11, 1946.

NORTHWESTERN PEDIATRIC SOCIETY

President: Dr. George B. Logan, 102 Second Ave. S. W., Rochester, Minn.
Secretary-Treasurer: Dr. Northrop Beach, 1009 Nicollet Ave., Minneapolis.
Place: Minneapolis, St. Paul, Duluth and Rochester. Time: January, April, July and October.

ROCKY MOUNTAIN PEDIATRIC SOCIETY

President: Dr. G. R. Fisher, 23 E. Pikes Peak Ave., Colorado Springs, Colo.
Secretary: Dr. Joseph H. Lyday, 1850 Gilpin St., Denver.

SOUTHERN MEDICAL ASSOCIATION, SECTION OF PEDIATRICS

Chairman: Dr. William Weston Jr., 1428 Lady St., Columbia, S. C.
Secretary: Dr. Angus McBryde, 604 W. Chapel Hill St., Durham, N. C.

STATE

Alabama Pediatric Society

President: Dr. Jerome C. Chapman, 2160 Highland Ave., Birmingham.
Secretary-Treasurer: Dr. Ruth Berrey, 2021–6th Ave. N., Birmingham.
Place: Birmingham. Time: September 1946.

Arizona Pediatric Society

President: Dr. Vivian Tappan, San Clemente, Tucson.
Secretary: Dr. Hilda Kroeger, Arizona State Health Dept. (Maternal and Child Welfare Division), Phoenix.
Place: Phoenix or Tucson. Time: At time of state meeting.

Arkansas State Pediatric Association

Chairman: Dr. C. B. Billingsley, 1425 N. 11th St., Fort Smith.
Secretary: Dr. R. E. Weddington, 1425 N. 11th St., Fort Smith.

California State Medical Society, Section on Pediatrics

Chairman: Dr. Charles W. Leach, 2000 Van Ness Ave., San Francisco 9.
Secretary: Dr. Chester I. Mead, 1930 Truxton Ave., Bakersfield.
Place: Biltmore Hotel, Los Angeles. Time: May 7-10, 1946.

Florida State Pediatric Society

President: Dr. Ludo Von Meysenbug, Box 3356, Daytona Beach.
Secretary: Dr. Robert Blessing, 409 Blount Bldg., Ft. Lauderdale.
Place: Concurrent with state association convention and fall meeting.

Georgia Pediatric Society

President: Dr. Ruskin King, 10 W. Taylor St., Savannah.
Secretary-Treasurer: Dr. Don F. Cathcart, 478 Peachtree St. N. E., Atlanta.
Place: Atlanta. Time: At time of annual state meeting.

Hezekiah Beardsley Pediatric Club of Connecticut

President: Dr. Edward T. Wakeman, 129 Whitney Ave., New Haven.
Secretary: Dr. Herman Yannet, Southbury Training School, Southbury.
Time: Two meetings a year.

Illinois State Medical Society, Section on Pediatrics

Chairman: Dr. Craig D. Butler, 715 Lake St., Oak Park.
Secretary: Dr. A. J. Fletcher, 139 N. Vermilion, Danville.

Indiana State Pediatric Society

President: Dr. K. T. Knode, 1105 E. Jefferson Bldg., South Bend.
Secretary-Treasurer: Dr. Matthew Winters, 621 Hume Mansur Bldg., Indianapolis.
Time: Two meetings a year.

Iowa Pediatric Society

President: Dr. Mark L. Floyd, Children's Hospital, Iowa City.
Secretary-Treasurer: Dr. James Dunn, Davenport Bank Bldg., Davenport.
Place: Des Moines. Time: April 18, 1946.

Medical Society of State of New York, Section on Pediatrics

Chairman: Dr. Carl H. Laws, 12 Pierrepont St., Brooklyn, N. Y.
Secretary: Dr. George R. Murphy, 292 Madison Ave., New York.
Place: Hotel Pennsylvania, New York. Time: May 2-3, 1946.

Medical Society of the State of Pennsylvania, Pediatric Section

Chairman: Dr. Elwood W. Stitzel, 403 Central Trust Bldg., Altoona, Pa.
Secretary: Dr. P. F. Lucchesi, Philadelphia Hospital, Philadelphia.

Michigan State Medical Society, Pediatric Section

Chairman: Dr. Donald J. Barnes, 3001 W. Grand Blvd., Detroit, Mich.
Secretary: Dr. Mark F. Osterlin, Central Michigan Children's Clinic, Traverse City.
Place: Detroit. Time: September 1945.

Mississippi State Pediatric Society

President: Dr. Harvey F. Garrison Jr., 315 E. Capitol Pl., Jackson.
Secretary: Dr. Guy Verner, 126 N. Congress St., Jackson.

Nebraska Pediatric Society

President: Dr. John M. Thomas, 301 N. 49th St., Omaha.
Secretary-Treasurer: Dr. Gerald C. O'Neil, 640 City National Bank Bldg., Omaha.
Place: As announced by committee. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

North Carolina Pediatric Society

President: Dr. Arthur H. London Jr., 1105 W. Main St., Durham.
Secretary: Dr. Jay M. Arena, Duke Hospital, Durham.
Place: Variable. Time: Sept. 9, 1946.

Northern California Pediatric Society

President: Dr. John Mott Rector, 2000 Van Ness Ave., San Francisco.
Secretary: Dr. George Bates, 490 Post St., San Francisco 2.
Place: San Francisco. Time: Second Thursday of February, April, September and November.

Oklahoma State Pediatric Society

President: Dr. G. R. Russell, 604 S. Cincinnati Ave., Tulsa.
Secretary: Dr. Luverne Hays, 108 W. 6th St., Tulsa.
Place: With Oklahoma State Medical Association. Time: Annual Meeting.

South Carolina Pediatric Society

President: Dr. Lonita Boggs, 300 E. Coffee St., Greenville.
Secretary-Treasurer: Dr. Hilla Sheriff, 429 Wade Hampton Office Bldg., Columbia 10.
Place: Poinsett Hotel, Greenville. Time: May 2, 1946.

Texas Pediatric Society

President: Dr. Max Woodward, 202 N. Travis St., Sherman.
Secretary-Treasurer: Dr. John E. Ashby, 3610 Fairmount Ave., Dallas.
Place: Fort Worth. Time: October 1946.

Virginia Pediatric Society

President: Dr. Emily Gardner, 1100 W. Franklin St., Richmond 20.
Secretary: Dr. Louise Galvin, 214 S. Boulevard, Richmond 21.

WEST VIRGINIA STATE MEDICAL SOCIETY, SECTION ON PEDIATRICS

President: Dr. Andrew Amick, 1021 Quarrier St., Charleston.
Secretary: Dr. A. A. Shawkey, Professional Bldg., Charleston.
Place: Huntington. Time: May 13-15, 1946.

LOCAL

ACADEMY OF MEDICINE OF CLEVELAND, PEDIATRIC SECTION

Chairman: Dr. Charles L. Ruggles, 10515 Carnegie Ave., Cleveland.
Secretary: Dr. Walter Heymann, 2103 Adelbert Rd., Cleveland.
Place: Cleveland Medical Library Bldg. Time: 8:15 p. m., October, December, February and April.

ACADEMY OF MEDICINE, TORONTO, SECTION OF PEDIATRICS

President: Dr. John M. Ross, 530 St. Claire Ave. W., Toronto, Canada.
Secretary: Dr. J. H. Ebbs, 13 Queen's Park, Toronto, Canada.
Place: Academy of Medicine. Time: Third Thursday of each month.

BRONX PEDIATRIC SOCIETY

President: Dr. Jacob H. Turner, 121 W. 197th St., New York.
Secretary: Dr. Harry S. Pizer, 1726 Davidson Ave., New York.
Place: Concourse Plaza Hotel, 161st St. and Grand Concourse. Time: Second Wednesday of each month, except June, July, August and September.

BROOKLYN ACADEMY OF PEDIATRICS

President: Dr. Lambert Krahulik, 14 Sidney Pl., Brooklyn.
Secretary: Dr. Joseph Battaglia, 713 Bushwick Ave., Brooklyn 21.
Place: Granada Hotel. Time: 8:45 p. m. fourth Wednesday of February, March, April, May and November.

BUFFALO PEDIATRIC SOCIETY

President: Dr. A. Wilmot Jacobsen, 187 Bryant St., Buffalo, N. Y.
Secretary: Dr. Richard A. Downey, 786 Forest Ave., Buffalo 9, N. Y.
Place: Children's Hospital, 219 Bryant St. Time: 8:30 p. m., first Monday of each month from September to June.

CENTRAL NEW YORK PEDIATRIC CLUB

President: Dr. Arthur W. Benson, 2 St. Paul St., Troy.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Places: Various cities in New York. Time: Third Tuesday of April and October.

CHICAGO PEDIATRIC SOCIETY

President: Dr. Louis W. Sauer, 636 Church St., Evanston, Ill.
Secretary: Dr. Henry G. Poncher, 1819 W. Polk St., Chicago 12.
Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month, October to May inclusive.

CINCINNATI PEDIATRIC SOCIETY

President: Dr. Theodore K. Selkirk, 3605 Edwards Rd., Cincinnati.
Secretary: Dr. John H. Hunt, Kemper Lane Bldg., Cincinnati.
Place: Children's Hospital, Elland Ave., Cincinnati. Time: On call.

Dallas Pediatric Society

President: Dr. O. René Caillet, 4325 Cole Ave., Dallas, Texas.
Secretary-Treasurer: Dorothy Wyvell, Bradford Memorial Hospital for Babies, Texas.
Place: Children's Medical Center. Time: 1 p. m., fourth Saturday and 8:30 p. m. the first Thursday of each month.

Detroit Pediatric Society

President: Dr. Philip J. Howard, 2977 W. Grand Blvd., Detroit, Mich.
Secretary: Dr. John C. Montgomery, 564 Fisher Bldg., Detroit 2, Mich.
Place: Variable. Time: Five times a year.

Houston Pediatric Society

President: Dr. Raymond Cohen, 2300 Caroline St., Houston, Texas.
Secretary: Dr. Betty Moody, 526 Richmond Rd., Houston, Texas.
Place: College Inn, Houston. Time: Fourth Monday of each month.

Kansas City (Missouri) Pediatric Society

President: Dr. Edwin H. Schorer, 1103 Grand Ave., Kansas City.
Secretary: Dr. H. E. Petersen, Kirkpatrick Bldg., St. Joseph, Mo.
Place: Kansas City General Hospital. Time: On call.

Los Angeles County Medical Association, Pediatric Section

President: Dr. Milo B. Brooks, 1033 Gayley Ave. W., Los Angeles.
Secretary-Treasurer: Dr. Elena Boder, 1830½ Lucille Ave., Los Angeles 26.
Place: Los Angeles County Medical Headquarters, 1925 Wilshire Blvd. Time: Second Monday of February, April, June, October and December.

Medical Society of the County of Kings and the Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Abraham M. Litvak, 1145 Eastern Parkway, Brooklyn.
Secretary: Dr. Harold Levy, 750 St. Marks Ave., Brooklyn.
Place: 1313 Bedford Ave., Brooklyn. Time: 9:00 p. m. fourth Monday of each month.

Medical Society of the County of Queens, Inc., Section on Pediatrics

Chairman: Dr. Meyeron Coe, 217-02–91st Ave., Queens Village, N. Y.
Secretary-Treasurer: Dr. Edith A. Mittell, 144–38th Ave., Flushing, N. Y.
Place: Queens County Medical Bldg., Forest Hills, N. Y. Time: Third Monday of October, January, March and May.

Medical Society of the District of Columbia, Section on Pediatrics

President: Dr. Harry A. Spigel, 2647 Connecticut Ave., Washington, D. C.
Secretary-Treasurer: Dr. Perry W. Gard, 2520 Woodley Rd., Washington, D. C.
Place: Medical Society Bldg., 1718 M St. N. W. Time: 8 p. m., fourth Thursday of every month.

Memphis Pediatric Society

President: Dr. Kinsey M. Buck, 1193 Madison Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. Barton Etter, 1024 Madison Ave., Memphis, Tenn.
Place: Memphis Country Club. Time: 8 p. m., Dec. 13, 1946.

Milwaukee Pediatric Society

President: Dr. G. H. Fallman, 231 W. Wisconsin Ave., Milwaukee.

Secretary-Treasurer: Dr. R. P. Showalter, 411 E. Mason St., Milwaukee 2.

Place: Milwaukee Children's Hospital. Time: Second Wednesday in February, April, June, October and December.

New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. Alfred E. Fischer, 73 E. 90th St., New York.

Secretary: Dr. Robert O. Du Bois, 122 E. 76th St., New York.

Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

Oklahoma City Pediatric Society

President: Dr. William M. Taylor, 625½ N. W. 10th St., Oklahoma City.

Secretary: Dr. Jeanne McKinnon-Shotstall, 625½ N. W. 10th St., Oklahoma City.

Place: Oklahoma Club. Time: Third Thursday of each month.

Philadelphia Pediatric Society

President: Dr. Ruth Hartley Weaver, 1719 Rittenhouse St., Philadelphia.

Secretary: Dr. Sherman Little, 1740 Bainbridge St., Philadelphia 46.

Place: College of Physicians, 19 S. 22d St. Time: 8:30 p. m., second Tuesday in October, December, February and May.

Pittsburgh Pediatric Society

President: Dr. Elwood Stitzel, 403 Central Trust Bldg., Altoona, Pa.

Secretary-Treasurer: Dr. C. J. Stoecklein, Medical Arts Bldg., Pittsburgh 13.

Place: Pittsburgh Academy of Medicine. Time: Second Friday in October, December, February and April.

Richmond Pediatric Society

President: Dr. T. Stanley Meade, 913 Floyd Ave., Richmond, Va.

Secretary-Treasurer: Dr. Edwin L. Kendig Jr., 828 W. Franklin St., Richmond 20, Va.

Place: Medical College Hospital. Time: 8 p. m., third Thursday of each month, except June, July and August.

Rochester Pediatric Society

President: Dr. William L. Bradford, 260 Crittenden Blvd., Rochester 7, N. Y.

Secretary-Treasurer: Dr. Donald D. Posson, 222 Chili Ave., Rochester 11, N. Y.

Place: Rochester Academy of Medicine or arrangement by program committee. Time: Third Friday of each month from October to May.

St. Louis Pediatric Society

President: Dr. Joseph C. Jaudon, 337 N. Euclid Ave., St. Louis.

Secretary-Treasurer: Dr. Gilbert B. Forbes, 500 S. Kingshighway, St. Louis 10.

Time: Second Thursday of each month from October to May, inclusive.

Seattle Pediatric Society

President: Dr. D. M. Dayton, 740 St. Helens Ave., Tacoma.

Secretary: Dr. W. B. Chesley, 1306 Madison St., Seattle 4.

Place: College Club. Time: Third Friday of each month from September to June at 6:30 p. m.

Southwestern Pediatric Society

President: Dr. William Belford, 420 Walnut St., San Diego, Calif.

Secretary: Dr. Robert J. Dostal, 710 Wilshire Blvd., Santa Monica, Calif.

Place: University Club of Los Angeles. Time: 6:30 p. m., first Wednesday in January, March, May, September and November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Park Bradshaw, Muskegon, Mich.

Secretary: Dr. Harry A. Towsley, University of Michigan, Department of Pediatrics and Communicable Diseases, Ann Arbor, Mich.

Westchester County Medical Society, Pediatrics Section (New York)

President: Dr. John B. Ahouse, 27 Ludlow St., Yonkers, N. Y.

Secretary-Treasurer: Dr. Elvira Ostlund, 64 Highland Rd., Rye, N. Y.

Place: Hospitals of Westchester County. Time: Third Thursday in November, January, March and May.